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CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEOESOPHAGEAL FISTULA

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OF ALL operable congenital deformities seen in the newborn infant, none is associated with more difficulties than congenital atresia of the esophagus with tracheoesophageal fistula. The infant is usually underweight and not infrequently premature. Other congenital deformities may be incompatible with life or may be of such a nature that multiple early operations are necessary. By the time a diagnosis is made many of these infants have pneumonia due to aspiration of saliva and liquids, or atelectasis as a result of occlusion of the bronchi. Such an infant, too weak to cough, and too feeble to cry, is a poor subject for a two to three hour anesthesia. The operative repair is long and tedious and the postoperative course is made stormy by all sorts of local and general complications. It is little wonder that the mortality continues to be rather appalling.

Leven[1] and Ladd,[2] in 1938, each saved the life of a child with tracheoesophageal fistula by tying off the distal end of the esophagus and exteriorizing the upper end, but to Haight and Towsley[3] goes the honor of being the first to close a tracheoesophageal fistula and to suture the ends of the esophagus together successfully. Since that time the continued work of Haight,[4] Ladd and Swenson,[5] Leven,[6] Gross and Scott,[7] Swenson,[8] Bigger,[9] and many others has added much to the successful care of these infants.

DIAGNOSIS

The diagnosis of tracheoesophageal fistula could so easily be made if at the time of delivery the possibility of such a deformity was considered in all newborn children who breathe poorly immediately after birth, have excessive

From The Children's Memorial Hospital.
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mucus in the mouth and throat, and have cyanotic spells during the early hours after delivery. Unsuccessful attempts to introduce a catheter into the esophagus of such a child at once should arouse suspicion of congenital obstruction. Confirmation of the deformity is promptly established by introduction of a couple of cubic centimeters of Lipiodol through a catheter into the blind pouch and taking an x-ray picture. Ideally the diagnosis should be made before attempts at feeding result in regurgitation and aspiration.

Visualization of a blind upper end of the esophagus and the presence of air in the stomach or intestines makes a diagnosis of atresia of the esophagus *with* tracheoesophageal fistula (Fig. 1). However, if in the presence of atresia of the upper end of the esophagus, air is not seen in the gastrointestinal tract, a diagnosis is made of atresia of the esophagus *without* tracheoesophageal fistula. Occasionally the fistula between the distal esophagus and the trachea is so small that air does not pass through with ordinary respiration. In one such case a gastrostomy was done and feedings were begun. Pressure in the stomach forced milk upward through the fistula and caused fatal aspiration.

Fortunately, a congenital tracheoesophageal fistula is an uncommon deformity. Because it is uncommon the possibility of its existence is quite naturally overlooked.

ANALYSIS OF CASES

This review of 35 cases of atresia of the esophagus with and without tracheoesophageal fistula seen at the Children's Memorial Hospital during the past four years is presented, not as a proud display, but as a critical appraisal of the patient, its deformities, and their complications, of the methods of treatment, and of a possible simplification of the difficult technique of surgical repair.

The infant's weight at birth is of great prognostic significance. Nine of the 35 infants weighed less than 5 pounds on admission; 10 weighed between 5 and 6 pounds; 9 weighed between 6 and 7 pounds; and only 7 weighed 7 pounds or more. Furthermore, 4 of the infants were from two to six weeks premature. In 6 of the 9 infants weighing less than 5 pounds, the tracheoesophageal fistula was repaired and an end-to-end anastomosis of the esophagus performed. All died. Two of this group had extensive deformities incompatible with life and the ninth died following gastrostomy on the basis of a diagnosis of simple atresia of the esophagus. Later a fistula between the esophagus and trachea was demonstrated. The patient died after an attempt to close the esophagus transperitoneally.

Multiple deformities add to the hazard of operation. Besides the deformity of the esophagus one infant had a medulloblastoma of the cerebellum and two infants had subdural hematomas, all unsuspected, but found at post-mortem examination. Two infants had atresia of the rectum; one had bilateral extensive anomalies of the genitourinary tract; one had an interauricular septal defect, and one had a deformity of the ear and partial paralysis of the face.

The average age upon admission to the hospital was 3.4 days; the average age at operation was 4.1 days. The majority had pneumonia and/or atelectasis at the time of operation.

Three of the 35 infants had congenital atresia of the esophagus without tracheoesophageal fistula. One of these patients who had an antethoracic placement of the stomach followed by intrathoracic transplantation is reported in the following article in this issue of the JOURNAL. The balance, 32, had the most common type III deformity, that is, atresia of the upper end of the esophagus and a fistulous communication of the lower end of the esophagus with the trachea at or near the carina. Two were inoperable because of multiple deformities and died shortly after admission. One originally diagnosed as simple atresia of the esophagus proved later to have a small communication with the trachea.

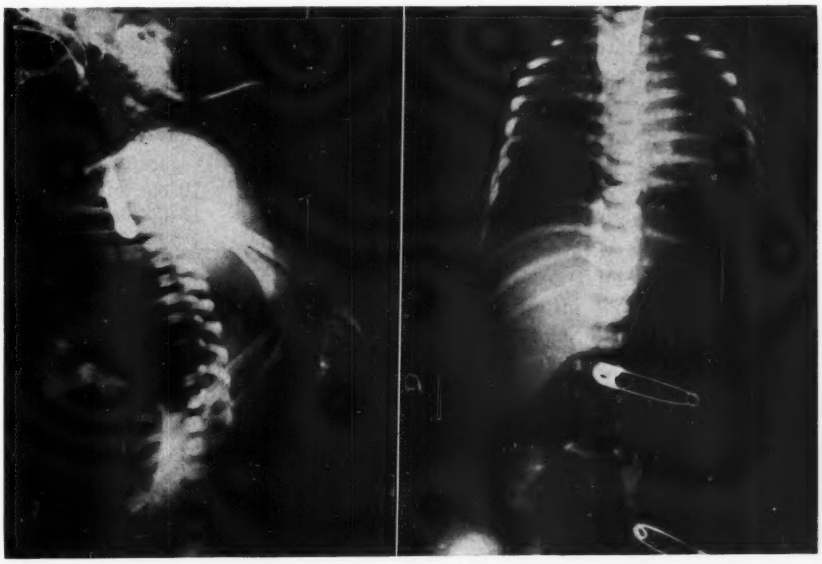

Fig. 1.—The blind upper end of the esophagus is filled with Lipiodol. Air in the stomach and intestine proves the presence of a tracheoesophageal communication.

Of the 29 patients with a classical type III deformity, primary anastomosis of the ends of the esophagus was done in 25. A preliminary gastrostomy followed by continuous suction as advised by Bigger was done in 3 patients to deflate the gastrointestinal tract and to allow time for treatment of the pneumonia. None survived this procedure. One child with associated congenital atresia of the rectum failed to survive a preliminary colostomy.

The 25 remaining patients with classical type III deformities are of primary interest. Closure of the tracheoesophageal fistula and reconstruction of the esophagus by end-to-end anastomosis was accomplished in all cases. Eleven of these patients survived the operation. One child died at 3 months of age because food would not go through the gastrointestinal tract. Exploratory laparotomy and later post-mortem examination failed to reveal the cause. Ten of the patients in this group are living and well.

PREOPERATIVE CARE

The preoperative care of these infants is demanding. Fluids are given with caution. If the child is dehydrated, sufficient 5 per cent glucose and small amounts of physiologic saline solution are given intravenously by needle or plastic tube in the saphenous vein at the ankle. While the average infant requires approximately 60 to 75 c.c. of fluid every twenty-four hours, per pound of body weight (15 c.c. per pound of this as saline solution), the infant with tracheoesophageal fistula is given about 40 to 50 c.c. as maintenance fluid requirement. Too much fluid to these patients, especially saline solution, may produce pulmonary edema.

On admission the child is immediately placed in oxygen. The pharynx is carefully aspirated with a soft rubber catheter about every half hour. Haight advised frequent turning of the infant and in case of atelectasis suggests placing the child on the opposite side with the head slightly lowered. Adequate doses of penicillin and streptomycin are immediately begun. Age and condition of the infant will determine the necessity for an immediate blood transfusion. During the operation approximately 10 c.c. of blood per pound of body weight is routinely given.

Bigger has made the excellent suggestion that gastrostomy be done before the tracheoesophageal fistula is attacked. Continuous suction on the gastrostomy tube not only deflates the gastrointestinal tract, but also prevents regurgitation of irritating gastric juice and bile into the bronchial tree. We have followed his advice in the treatment of 4 cases. The fact that 3 of the 4 died is due, not to any failure of the procedure, but to the extremely poor condition of the patients.

OPERATION

Anesthesia for these infants is a problem. We have used cyclopropane and oxygen administered through a tightly fitting face mask, or through an intratracheal tube. Recently we have been using open-mask ether. The last four patients operated upon have all had open-mask ether and all survived. No conclusions may be drawn from this sudden burst of good fortune. The small face mask is always handy for administration of pressure oxygen at a moment's notice. Likewise an intratracheal tube and laryngoscope are nearby for any emergency.

After the child is anesthetized, it is turned to the prone position with the right side slightly elevated and the right hand drawn upward (Fig. 2, *A*). Through a parascapular skin incision the rhomboid and trapezius muscles are cut and the scapula is retracted laterally. We resected subperiosteally segments of two, three, or four ribs until Leven suggested that instead we resect a long segment of one rib—the third or fourth. There is no doubt that resection of one rib is far superior to the removal of segments of a number of ribs. Exposure is better, there is less danger of tearing the pleura and the hazard of postoperative paradoxical movements of the chest is eliminated. The parietal pleura is carefully pressed away from the chest wall and vertebrae

with a spatula and a tiny piece of gauze caught in the end of fine hemostat. The only place where there is real danger of injuring the pleura is during ligation of the azygos vein. Haight avoids this pitfall by not cutting the azygos vein but merely pushing it aside. If perchance, a hole is made in the pleura it is at once repaired if possible, otherwise covered with a pledget of gauze. The lung beneath is collapsed and retracted with a broad malleable retractor.

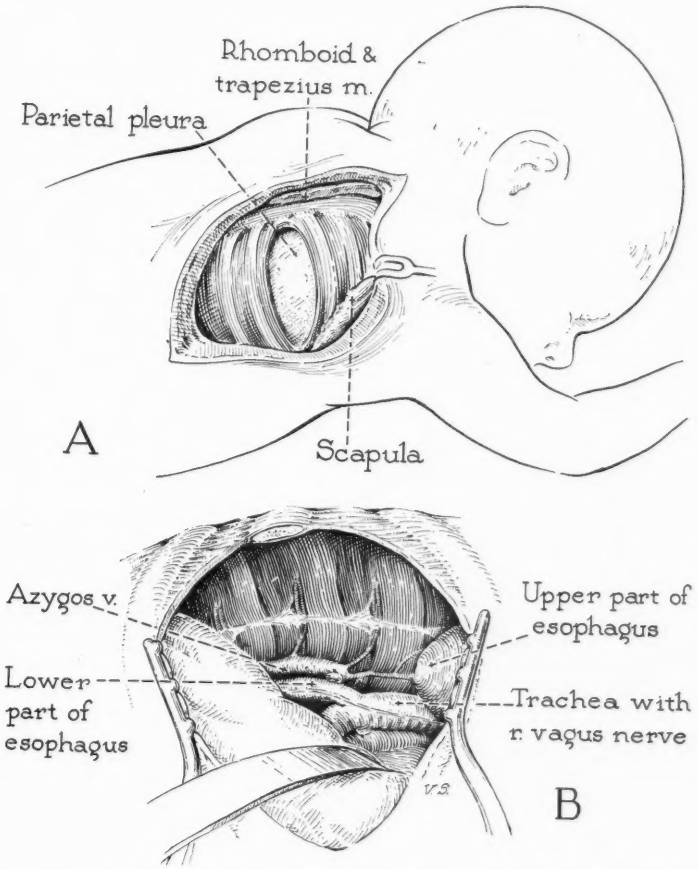

Fig. 2 *A* and *B*.—*A*, The fourth rib has been resected subperiosteally. *B*, The pathology is exposed.

The distal end of the esophagus is easily found and dissected free as far as necessary for proper handling. It is transfixed and tied with a silk suture near its attachment to the trachea. The esophagus is grasped with a fine-toothed clamp and cut near the transfixion suture. A few interrupted silk sutures are placed in the stump of the esophagus on the trachea (Fig. 2, *C*).

The upper end of the esophagus is caught at the very tip with an Allis forceps, drawn down, dissected from surrounding structures, and freed from its fibrous attachment to the trachea (Fig. 2, *C*). If there is the slightest difficulty in finding the upper end of the esophagus a catheter pushed down through the mouth will promptly identify it. A similar toothed clamp is now applied

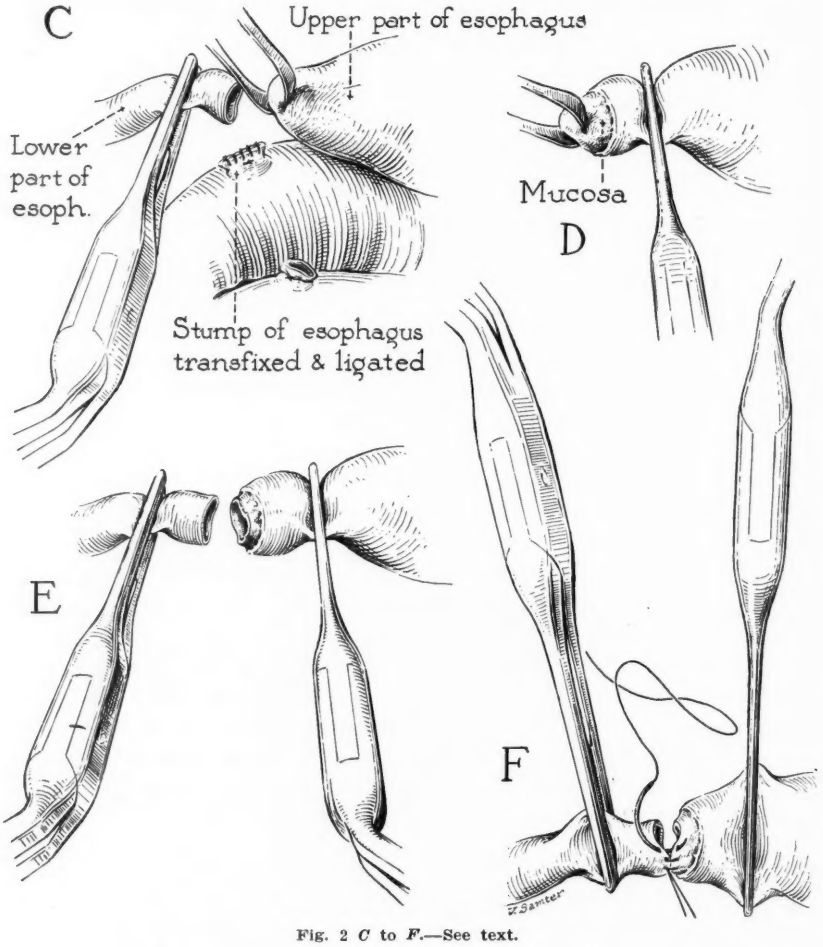

Fig. 2 *C* to *F*.—See text.

across the upper segment (angulated clamps are desirable because they allow more room for suture), and the small bit of muscularis caught in the Allis clamp is cut off. The muscularis is pushed up a bit and an opening to correspond to the size of the lumen of the lower segment is made in the mucosa (Fig. 2, *D*).

The ends of the esophagus are held together with the clamps. In the last six patients operated upon we have used the first models of the fine-toothed

ductus clamps previously described.[10] These clamps are somewhat lighter than those routinely used at present for division of a patent ductus arteriosus. The teeth interdigitate slightly. Tearing of the muscularis occurred in one case because a heavier coarctation forceps was used. It is extremely important that the clamp be completely closed or it will slip and tear the esophageal wall. As the ends of the esophagus are being brought together most of the traction should be exerted on the upper end which is thick and strong. We

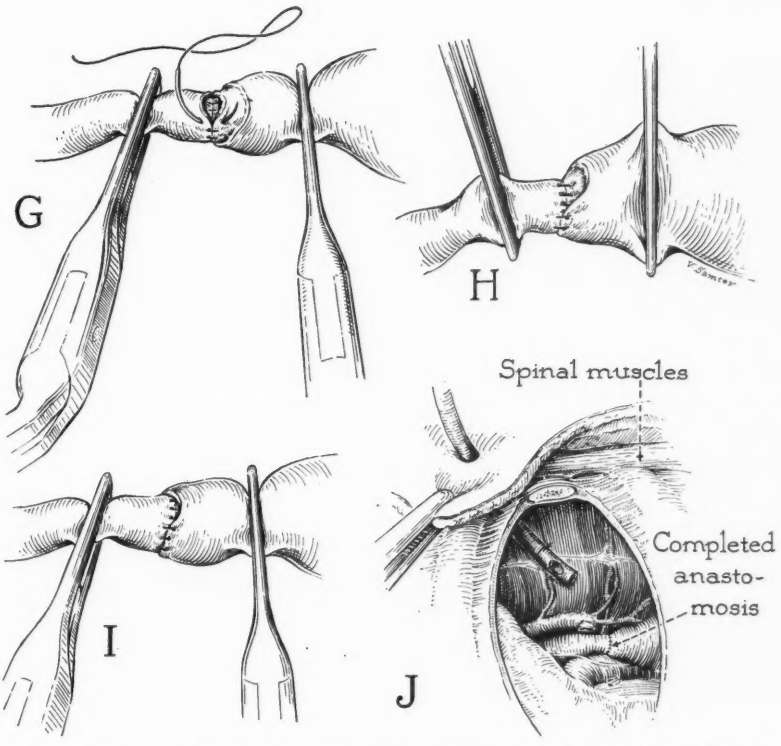

Fig. 2 *G, H, I,* and *J.*—*G, H,* and *I,* see text. *J,* The drainage tube is inserted through a small stab wound and is fixed to the fascia of the vertebrae.

have found the clamps superior to traction sutures which are likely to pull out and tear the esophagus. The telescoping technique of anastomosis advised by Haight is routinely used. With the clamps held straight up the posterior row of sutures between the mucosa of the upper segment and the entire thickness of the lower segment are placed (Fig. 2, *E* to *J*). Fine silk, 00000 Deknatel swaged on a small curved needle, has been used. We have recently used with satisfaction 000000 silk Deknatel on a smaller needle as advised by Haight. All the sutures are so placed that the knots are to the outside. After the posterior row of sutures has been placed the clamps are lowered and the anterior row is placed in a similar manner. The clamps are

again elevated and the muscularis is drawn down over the original line of suture and attached to the wall of the lower segment. Once more the clamps are lowered and the anastomosis is completed anteriorly in a similar manner.

Drainage of the mediastinum is absolutely necessary. Until Bigger published his paper on this subject in 1949, we routinely drained through the incision. Two patients died of collapse of the lung and tension pneumothorax outside of the pleura due to air aspirated through the wound presumably along the drainage material. Bigger's most valuable suggestion is that a drainage catheter be inserted through a separate small stab wound in an interspace below the incision near the edge of the vertebra (Fig. 2, *J*). The No. 10 or 12 French catheter with a couple of extra holes near the end is drawn through the stab wound into which it fits very tightly and is brought to lie near, but not against, the anastomosis. Pressure on the lung by the tip of the catheter can be avoided by throwing a fine catgut stitch around the catheter and suturing it to the fascia over the vertebra.

The lung is now expanded by adjusting the anesthetic mask to the child's face and making pressure on the anesthetic bag. After the lung has been completely expanded the chest is closed snugly by suturing the adjoining ribs together with a running suture of surgical gut. The wound is closed in layers with surgical gut and the skin with silk. While the anesthetist is making pressure on the anesthetic bag a clamp is applied to the drainage catheter to maintain expansion of the lung.

POSTOPERATIVE CARE

The infant is returned to an oxygenated crib. The drainage catheter is connected with a constant suction machine to remove any fluid, but above all to keep the lung expanded. To avoid excessive negative suction a water bottle with an escape tube, which allows only 10 cm. of water negative pressure, is connected in the suction system. The tube is left in the chest for at least ten days; suction is maintained until it is sure that the pleura is fixed.

Penicillin and streptomycin are continued. Fluids in the form of glucose, plasma, and occasionally blood, are given as necessary. Sodium chloride solution is given in very small amounts. The child is kept in oxygen a number of days. If mucus tends to collect in the throat, careful aspiration is done by a trained person who will not push the catheter down the recently sutured esophagus. Aspiration of the throat often stimulates a beneficial cough.

Until recently gastrostomy was routinely done forty-eight hours after repair of the esophagus. It is always done if leakage from the anastomosis is profuse. A gastrostomy in such cases is a necessary evil, the source of annoying and often disastrous complications. If there is no leakage, or if it is very slight, a gastrostomy now is not done. On about the third day the infant is given a few cubic centimeters of water every hour. The child is swallowing saliva anyway—it might as well swallow a little sterile water to help keep the esophagus clean. If all goes well the water is slowly replaced by a weak formula cautiously increased to a maintenance diet. In the presence of marked leakage, feeding by gastrostomy must be done by a slow continuous drip or the feedings will promptly flow out of the posterior wound.

Postoperative stricture of the esophagus at the point of anastomosis is expected if leakage has been profuse. To facilitate dilatation, it is desirable to have a string through the esophagus but to get it there may be difficult. The infant is given a fine thread by mouth and some formula to wash it down. If the thread does not come out of the back it is assumed that it is in the stomach. The stomach is filled with water and the gastrostomy tube is promptly removed. The thread floats out of the gastrostomy opening with the water—sometimes. A heavy string is then tied to the thread and drawn

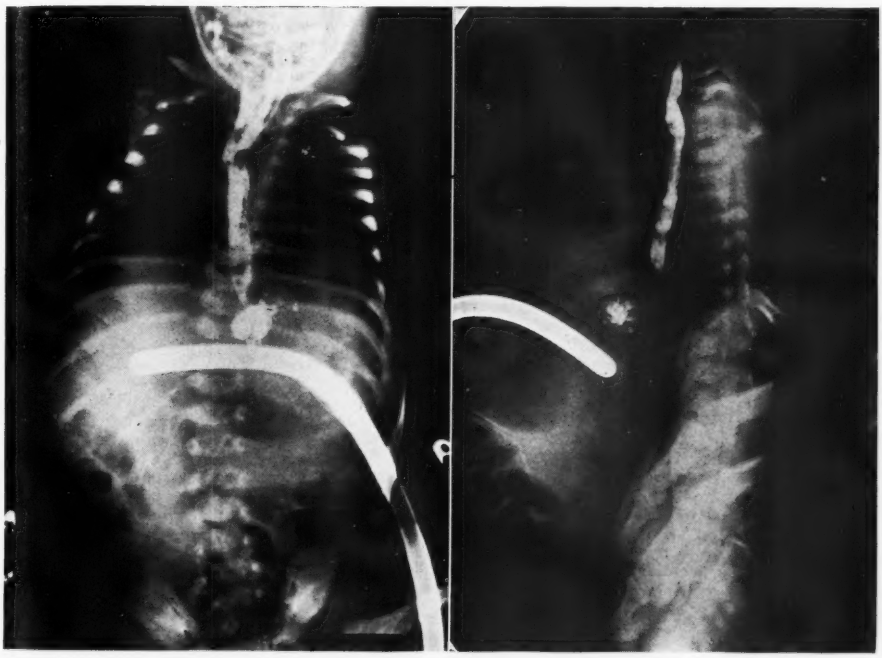

Fig. 3.—Anteroposterior and lateral roentgenographic views of an esophagus two weeks after anastomosis. No stricture developed.

through the esophagus and the ends of the string are tied together. If the thread will not go down, a fine catheter is passed through the esophagus into the stomach under the fluoroscope. The catheter is fished out of the stomach and the string tied to its other end is drawn through the esophagus. From three to thirteen dilatations were necessary because of varying degrees of stricture in five children. The gastrostomy is kept open until it is demonstrated that no further dilatations are necessary. Dr. Paul Holinger and Dr. Albert Andrews, of the Endoscopic Department, prefer, when possible, to do retrograde dilatations of esophageal strictures with graduated bougies attached to a string and drawn through the esophagus.

Postoperative roentgenograms of the esophagus following a swallow of barium (Fig. 3) may appear discouraging but should not be interpreted too harshly. In spite of the deformity the child can usually swallow without difficulty. However, optimism should be controlled or a seemingly harmless deformity may suddenly terminate in total stricture very difficult to open.

SUMMARY

Thirty-five patients with congenital atresia of the esophagus with or without tracheoesophageal fistula are analyzed, not to justify, but explain the continued high mortality associated with surgical correction of these deformities. In 25 infants the fistula was closed and the ends of the esophagus were anastomosed. Eleven patients survived the operation and 10 are thriving and well from three months to three years following operation. Holding the ends of the esophagus together with fine-toothed clamps while the anastomosis is being performed appears advantageous in simplifying the technique of suture.

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(See discussion on page 701)

CONGENITAL ATRESIA OF THE ESOPHAGUS

Antethoracic Placement of the Stomach Followed by Intrathoracic Transplantation

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WHEN congenital atresia of the esophagus is associated with tracheoesophageal fistula, closure of the fistula and end-to-end anastomosis of the segments of the esophagus are usually possible. (See preceding article in this issue of the Journal.) However, in 3 infants who had congenital atresia of esophagus *without* communication with the trachea, the distal segment was too small and too short to be brought in contact with the upper segment. One of these children, 6 days old on admission, died two days after simple gastrostomy. An unsuccessful attempt was made in the second child to bring the widely separated blind ends of the esophagus together. Following gastrostomy and exteriorization of the upper end of the esophagus, the child did well for one year. Antethoracic placement of the stomach was then followed in a few hours by death from hyperpyrexia although the operation was simple technically. The third case, reported in detail, is of interest because of unanticipated difficulties corrected by a surgical procedure hitherto undescribed.

CASE REPORT

Marie H., aged 2 days, weighing 5 pounds and 3 ounces, was brought to the Children's Memorial Hospital in an oxygen cabinet, Dec. 12, 1947. At birth much fluid was aspirated from the throat before normal respiration was established. The puny, wrinkled infant was too feeble to cry. Thick mucus constantly collected about the baby's mouth. Injection of 2 c.c. of Lipiodol through a catheter into the upper end of the esophagus revealed atresia. Absence of air in the gastrointestinal tract indicated atresia without communication with the trachea (Fig. 1). The lungs were relatively clear. General physical examination revealed no other deformities. The child was kept in oxygen for twenty-four hours and given sufficient fluid in the form of 5 per cent glucose and saline solutions to meet physiologic requirements. The pharynx was aspirated each half hour.

On December 13, under closed mask cyclopropane anesthesia, through a right parascapular incision segments of the second to fifth ribs were resected subperiosteally. Extrapleural dissection of the mediastinal region revealed that the distal segment of the esophagus was short and very small. In fact, at rest the segments of the esophagus were separated by almost three inches and could not be brought together by traction. The chest wall was closed without drainage. During the operation 50 c.c. of whole blood were given by needle in the saphenous vein at the ankle.

The following day the resident correctly and frankly noted on the chart, "the child looks surprisingly good." She was kept in oxygen and frequent suction of the pharynx was continued. Weight fell to 4 pounds and 11 ounces.

On December 15, under general anesthesia a gastrostomy was done. The stomach was exposed through a muscle splitting incision in the right upper quadrant and a de Pezzer catheter inserted through a small incision in the stomach wall. The stomach in-

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cision was tightly closed about the catheter and the abdominal wound was closed. Small feedings were cautiously begun and built up to maintenance by December 18 when the child was 8 days old. Because transplantation of the stomach into the chest with anastomosis of the fundus to the upper end of the esophagus inside the chest was contemplated, exteriorization of the upper end of the esophagus at the neck was delayed. In retrospect this delay was unwise because atelectasis developed. Furthermore, in view of the respiratory difficulty this child had at 22 months of age when the stomach was placed in the chest, I do not believe it would have tolerated such a procedure during early infancy. Nursing care of the infant was a tremendous burden because of the necessity of frequent pharyngeal aspirations of mucus.

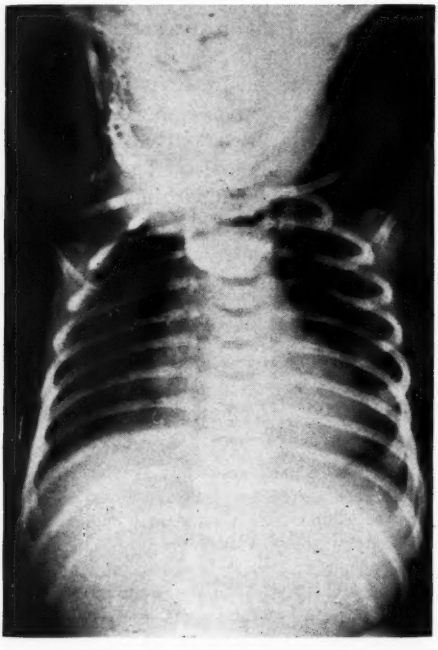

Fig. 1.—Absence of air in the gastrointestinal tract indicates atresia of the esophagus without tracheoesophageal fistula.

On December 31, the baby rather suddenly became cyanotic and showed dullness of the entire right chest. Roentgen examination revealed atelectasis. Prompt endoscopic aspiration of secretions in the bronchus improved the child's condition considerably. Repetition the following day was followed by return of breath sounds in the right chest and return of the heart to the left side.

On Jan. 16, 1948, the upper end of the esophagus was exteriorized above the left clavicle under cyclopropane anesthesia. The infant now improved rather promptly and was discharged from the hospital February 4, weighing 7 pounds and 7 ounces. Gastrostomy feedings were continued at home. On Sept. 28, 1948, at the age of 10 months the child, now weighing 15 pounds, 6 ounces, was readmitted to the hospital. About this time attention was called to an article by Rienhoff[1] describing antethoracic transplantation of the stomach and anastomosis of its fundus to the exteriorized cervical esophagus. Rienhoff, in a personal communication, vouched for the simplicity and nonshocking quality of this procedure. His predictions were substantiated.

On October 4, under ether anesthesia, a curved left rectus incision was made in the upper abdomen. The gastrostomy opening in the abdominal wall was freed and the gastrostomy closed with two layers of interrupted sutures placed parallel with the long axis of the stomach.

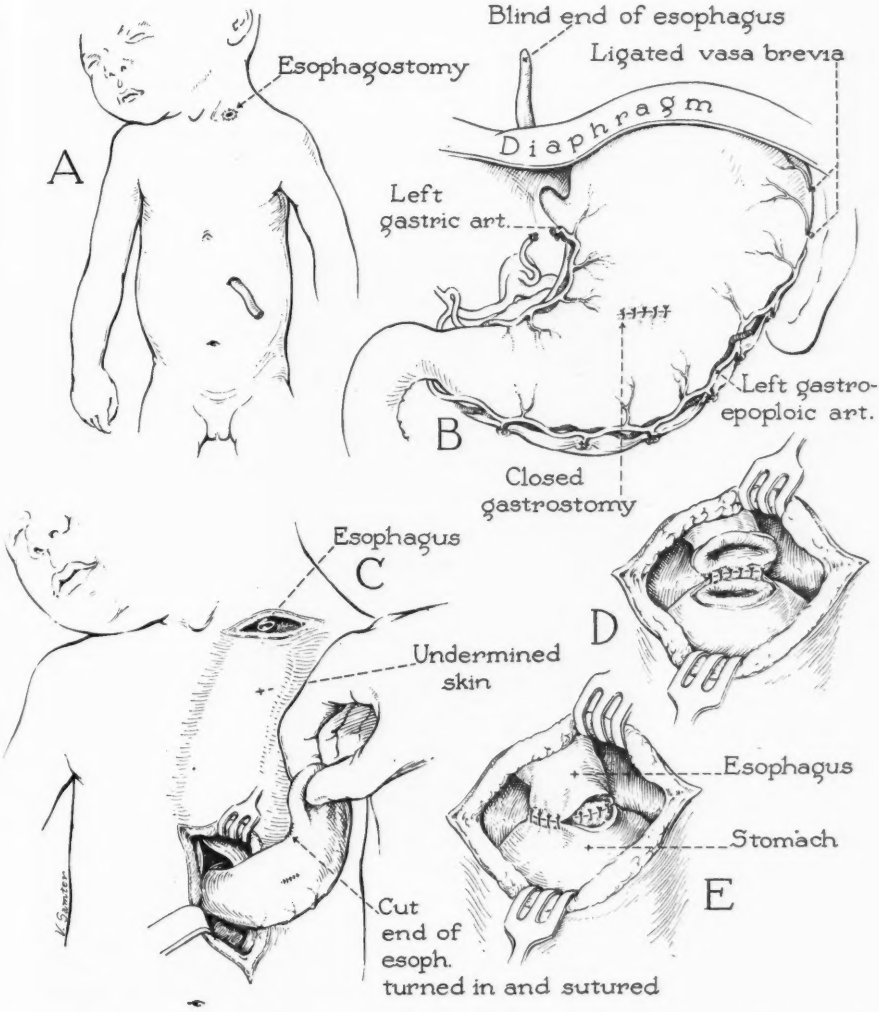

Fig. 2.—The stomach was freed, transplanted antethoracically, and its fundus anastomosed to the cervical stump of the esophagus.

The transverse colon was separated from the greater curvature of the stomach. The vasa brevia and the left gastric artery were doubly ligated and cut (Fig. 2). The lower segment of the esophagus was drawn out of the mediastinum with considerable difficulty but without accident. The esophageal stump was cut off at its point of entrance into the

stomach and the opening closed with two rows of fine silk sutures parallel with the long axis of the stomach. The stomach was entirely freed.

A transverse incision was made above the left clavicle with the esophagostomy wound in the center of the incision. The end of the cervical esophagus was dissected free from the surrounding structures. The skin over the left part of the sternum and the anterior margin of the adjacent ribs was freed by blunt dissection from the xiphoid to the line of incision above the clavicle. The fundus of the stomach was grasped with an Allis forceps and drawn beneath the skin to the end of the cervical esophagus. It was interesting to note that the stomach could be drawn to this position without tension, in fact, it could easily have been drawn up another inch. An anastomosis between the cervical end of the esophagus and the fundus of the stomach was accomplished with two layers of fine interrupted silk sutures. It was gratifying to see free bleeding from the incision in the fundus preparatory to the anastomosis. The cervical and abdominal wounds were closed. Technically the operation was simple and free from shock.

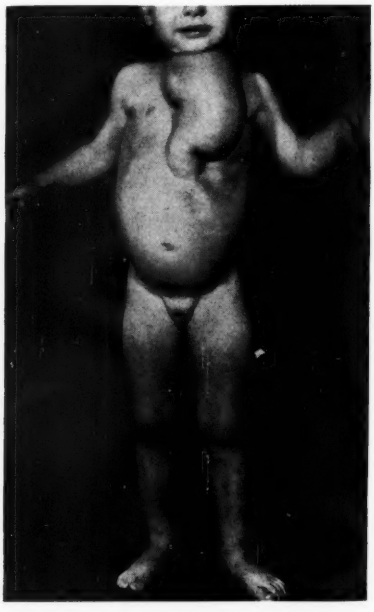

Fig. 3.—The subcutaneous stomach functioned well but was huge and unsightly.

The postoperative course was entirely uneventful except for a small stitch abscess at the outer angle of the neck wound. Four days after operation feeding was begun and by the tenth postoperative day a maintenance diet was being taken. A swallow of barium on the fourteenth day after operation went into and through the stomach without evidence of obstruction.

The child was dismissed from the hospital and in general progressed very well. Food was taken eagerly. The first few months after operation the subcutaneous stomach was not excessively large, but as months went by the stomach increased markedly in size (Fig. 3) although it functioned perfectly. After an ordinary meal the child's chin was literally buried in the fundus of its stomach. The parents were willing and eager that an attempt be made to correct the deformity.

Two methods of placing the stomach in the chest were considered; one, severing the esophagogastric anastomosis, freeing the stomach from its antethoracic position, drawing it through the diaphragm to the apex of the chest, and reanastomosing it to the esophagus. The idea of breaking down the anastomosis did not seem attractive. Consequently an alternative method not heretofore described was considered and tried in the post-mortem room. It appeared feasible.

On Sept. 28, 1949, the child, now 22 months old, was readmitted to the hospital and under intratracheal cyclopropane-oxygen anesthesia the following operation was performed. The stomach was thoroughly emptied. A cannula was placed in the saphenous vein at the ankle for administration of fluid and blood. A long skin incision was made from the left clavicle to a point midway between the xiphoid and the umbilicus. The stomach was easily separated from the skin and sternum. It had established no vascular communications with the surrounding tissue. The stomach was entirely freed from neck to the pylorus. The abdomen was opened sufficiently to expose the diaphragm.

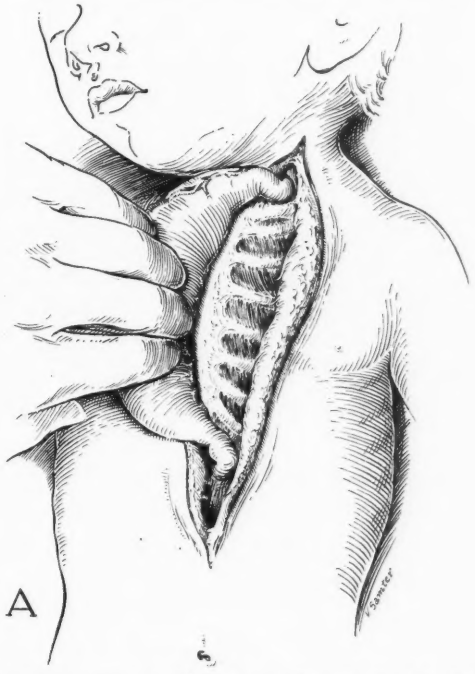

Fig. 4.—The stomach was freed and all the costal cartilages to the left of the sternum exposed.

Extensions of the fibers of the pectoralis major muscle were freed from the costal cartilages along the left edge of the sternum (Fig. 4). Beginning at the lower margin the costal cartilages, from the seventh to the second inclusive, were cut slightly lateral to the sternocostal junction. The intercostal arteries were tied as the ribs were cut. The pericardium was easily pushed away from the costal cartilages. As the upper ribs were cut the left pleural cavity was opened. The medial portion—approximately one-half—of the clavicle and a similar segment of the first rib were removed with the periosteum. The vessels beneath were pushed backward.

The diaphragm was now split straight back to the vertebrae. The ribs were retracted laterally, the stomach was placed in the left pleural cavity behind the lung and

the heart, and fixed to the pleura in this position (Fig. 5). The diaphragm was repaired allowing sufficient room for stomach emptying. A de Pezzer catheter was drawn through the seventh interspace from the inside out for drainage. The costal cartilages were apposed and fixed, each with a through-and-through silk suture. The fascia of the pectoralis major muscle was drawn over the line of incision in the ribs and sutured to the sternum thereby giving an airtight closure of the chest. The lung was re-expanded and the catheter drain clamped. The abdominal portion of the wound was closed. The skin edges were coapted with a running silk suture. The esophagus lay easily in the depression made by resection of portions of the first rib and clavicle. The operation required two and one-half hours. A transfusion of 250 c.c. of whole blood was given during operation. The child was returned to her room in excellent condition and placed in oxygen. The drainage tube was attached to an under-water seal.

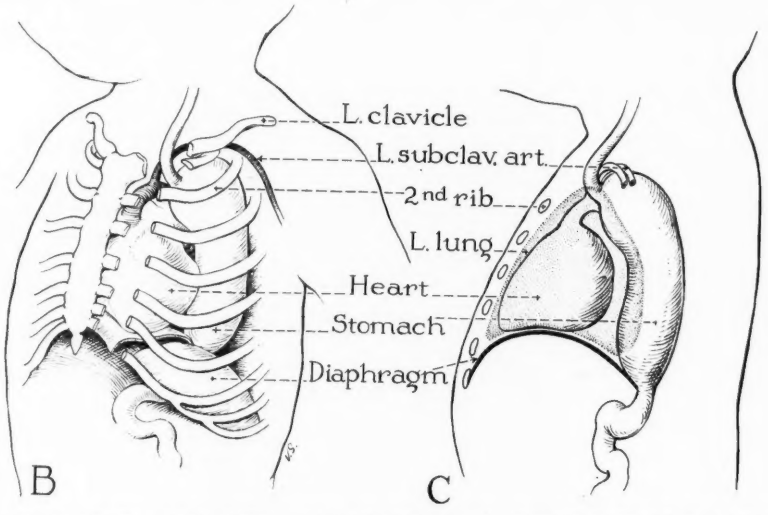

Fig. 5.—*B*, The costal cartilages were cut and segments of the first rib and clavicle were removed. The diaphragm was split.
C, The stomach was placed and fixed behind the lung.

For three days the child did very well and was started on a liquid diet including milk. The stomach became distended with air. Respirations became very rapid (70 to 80 per minute) and cyanosis was visible. For two days all food and fluids were refused. Her condition was precarious. A Levine tube was inserted transnasally into the stomach for feeding, but the child objected so strenuously that it was removed in twenty-four hours. Penicillin injections, intravenous fluids, and Levine tubes made the spirited child completely uncooperative. The mother, a very intelligent woman, was asked to come to the hospital to feed and care for the child. Promptly the child began to take fluids and food willingly and on the fourteenth day after operation was discharged in very good condition but exceedingly suspicious of every man in white (Fig. 6).

During the first month after operation she gained a pound in weight. Now, six months after the final operation, she is doing very well, eats four meals a day and plays vigorously. Respirations were somewhat rapid for a time but are now normal.

It is appropriate to mention gratefully the tremendous amount of time and care given this patient by the nursing and resident staff. Such children require almost constant nursing service. This child received approximately 600 injections of penicillin and streptomycin, 13,000,000 units of the former and 8 million units of the latter during

the total hospitalization of 123 days. The residents administered 59 flasks of intravenous solutions, such as: blood, plasma, glucose, saline, and Amigen. Most of it was given intravenously. Every available vein in the legs, arms, and scalp was used to capacity.

DISCUSSION

Every effort should be and is being made by surgeons to bring together the ends of a congenitally atretic esophagus. At times because of wide separation of the segments, anastomosis is impossible and gastrostomy, plus exteriorization of the upper end of the esophagus, is necessary.

Fig. 6.—Photograph of child ten days after operation.

Various procedures have been devised to overcome the misfortune of a permanent gastrostomy. Ladd[2] had some brilliant successes and a number of failures in the construction of an antethoracic esophagus of skin tubes. Unfortunately, regurgitated gastric juice breaks down a skin tube esophagus and results in fistula formation. Swenson[3] in a number of cases constructed an esophagus of jejunum which he placed antethoracically beneath the skin and anastomosed to the exteriorized cervical esophagus. In this procedure, since the stomach is by-passed, irritating gastric juices cannot reflux and produce fistulas. He had difficulty in some cases in bringing the jejunum to the esophagus and had to insert a short skin tube. Harrison[4] has brilliantly accomplished the construction of an *intrathoracic* esophagus in two cases from a segment of jejunum. This operation, done in three stages, has the decided advantage of avoiding the cosmetic defect of any type of subcutaneous esophagus. Anastomosis of the distal end of the transplanted jejunum to the stomach, as done by Harrison, is also desirable. Sweet[5] successfully restored continuity of the alimentary canal by transplanting the stomach of a 21-month-old child into the chest and anastomosing the fundus to the cervical esophagus. Considering the distressing and alarming dyspnea our patient at age 22 months had for

a few days after intrathoracic placement of the stomach, it is doubtful whether such an operation should be done during early infancy when encroachment on respiratory space is poorly tolerated. All methods devised for restoration of a normal food passage are worthy of consideration and development. Failures and a high operative mortality should not discourage surgeons from making strenuous efforts to relieve a child of the distressing burden of a permanent gastrostomy.

CONCLUSION

Although the case of this child terminated happily, antethoracic transplantation of the stomach is not advised.

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(See discussion on page 701)

RECONSTRUCTION OF ESOPHAGUS

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RECONSTRUCTION of the entire esophagus with a transthoracic jejunal transplant was first reported by Dr. Albert Harrison[1] at the meeting of this Association in Quebec in June, 1948. His work was based in turn upon that of Rienhoff,[2] Yudin,[3] Herzen,[4] and Roux.[5] Rienhoff was the first to transplant the jejunum within the thorax and has described fully the historical development of the operation in his article.[2]

Harrison performed his operation in three stages. At the first stage a tube of jejunum was prepared with one arcade of blood supply, isolated, brought up through the transverse mesocolon, and the distal end anastomosed to the stomach. Jejunal continuity was restored by end-to-end anastomosis. Two weeks later the left chest and diaphragm were opened and the esophagus divided at the cardia, freed, and brought out through an incision in the neck along with the proximal end of the jejunal tube. Finally, two weeks later the esophagojejunal anastomosis was made in the neck.

While listening to Dr. Harrison's excellent presentation, one was impressed with the magnitude of his procedure. The second stage of the operation seemed relatively shocking and freeing the transplanted loop of jejunum without injuring the blood supply appeared difficult. This feeling was confirmed during the discussion when Dr. Sweet stated that "the operation is not an easy thing to accomplish."[6]

A few weeks later, with these thoughts still in mind, a thymectomy was performed for myasthenia gravis. The ease and safety of dissection in the anterior mediastinum suggested a new location for the transplanted jejunum. It appeared that a relatively short tunnel could be made in the anterior mediastinum, safely, and, with a minimum of shock. A one-stage operation might be possible and many difficulties avoided.

Dissection on cadavers confirmed many of these suppositions. It was found that the internal mammary arteries were protected by the transverse thoracic muscles and the insertion of the sternothyroids (Fig. 1). If the anterior mediastinum is approached from above and below exactly in the midline, the dissecting fingers can press close to the under surface of the sternum and costal cartilages and, remaining safely in the plane of the endothoracic fascia, push back the parietal pericardium and parietal pleura to form a tunnel from abdomen to neck large enough to admit a hand (Fig. 2).

As in Dr. Harrison's cases, reconstruction of the entire esophagus was desired most often for lye strictures, but one hesitated to attempt an untried

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procedure on these patients. So it was first used as a palliative for inoperable carcinoma of the esophagus.

The technique changed as complications were encountered. Rienhoff's method of preparing an antiperistaltic tube of jejunum (Fig. 3) was used at first, but later abandoned reluctantly as the importance of the direction of

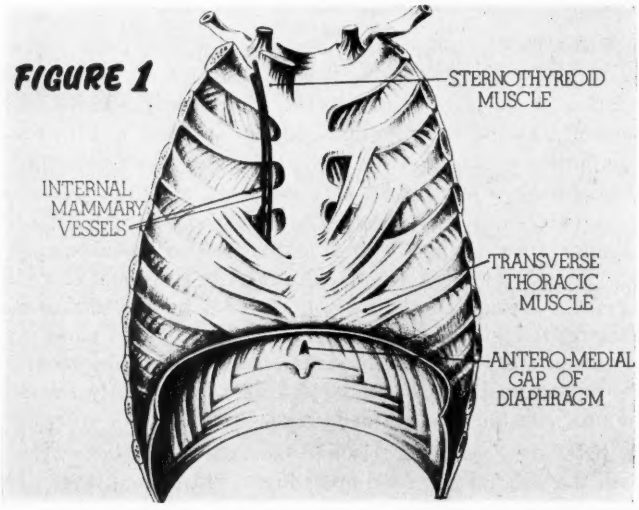


Fig. 1.—Anatomy of anterior chest wall viewed from behind.

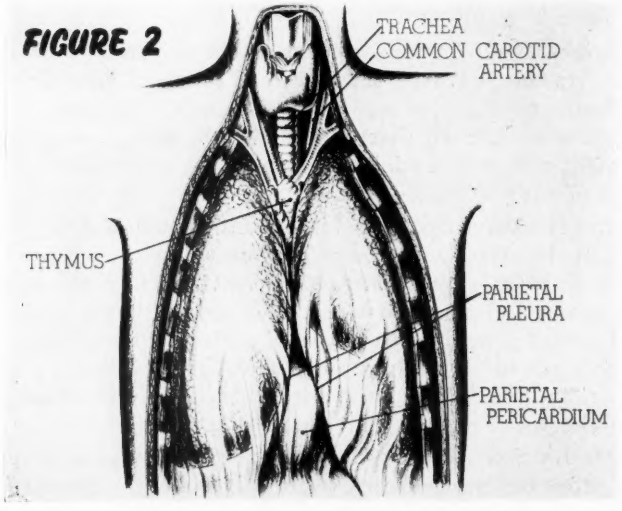


Fig. 2.—Anatomy of anterior mediastinum, the anterior chest wall having been removed.

peristalsis became evident. Our present technique as formed by the pressure of experience is as follows.

PRESENT TECHNIQUE

The intra-abdominal part of the procedure is best carried out through a left paramedian rectus-displacing incision extending from one inch above the xiphi-sternal junction to one inch below the umbilicus. This incision allows easy retraction to the right

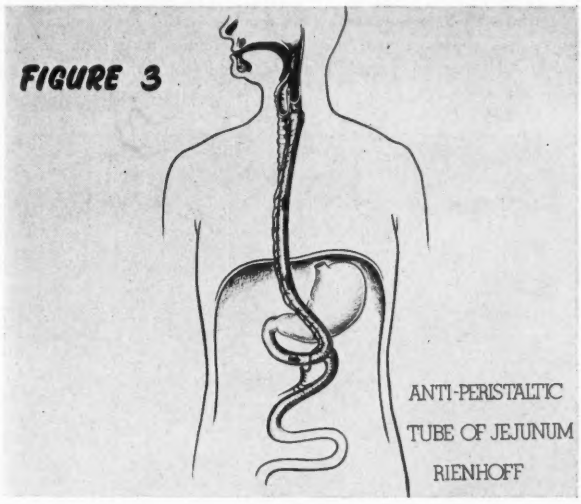


Fig. 3.

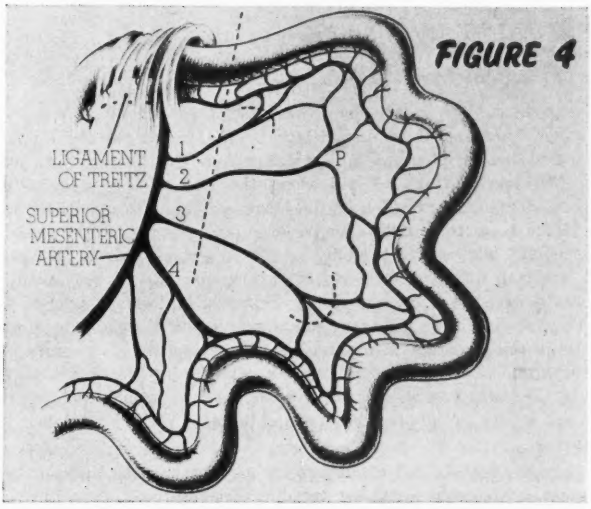


Fig 4.—Straight dotted line indicates sites for division of jejunum and vessels in preparing an isoperistaltic tube. Curved dotted lines indicates sites for division of secondary branches if additional length is required. The letter *P* indicates the preservation of secondary branches when the arcade appears dangerously thin.

of the ligamentum teres of the liver and, during the closure of the wound, facilitates the closing of the peritoneum around the jejunum as the latter passes from the peritoneal cavity to the anterior mediastinum.

The jejunum is brought out of the abdominal cavity, spread fan-wise on warm moist towels, and the distribution of the superior mesenteric artery studied (Fig. 4). The relative sizes of the first four or five branches should be noted. If the fourth or fifth branch is a large one, it means that the first three or four can be severed without jeopardizing the blood supply of the bowel, because the retrograde flow through the arterial arcade close to the intestine will be adequate. This can be tested by simultaneous digital compression of the vessels chosen for division. The jejunum may then be divided near the ligament of Treitz and therefore permit the distal end to be anastomosed to the esophagus in an isoperistaltic manner (Fig. 5).

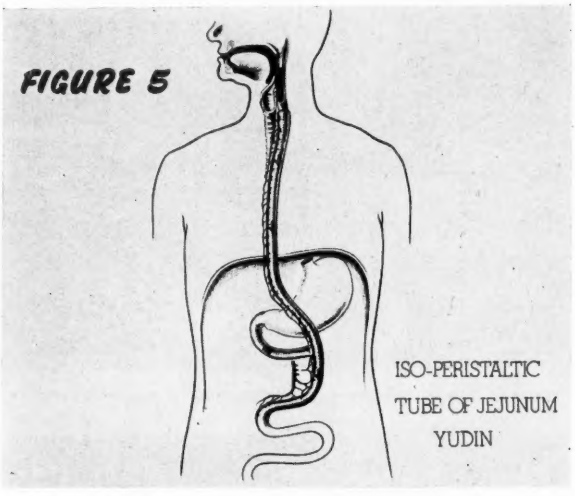


Fig. 5.

The distance from the thyroid cartilage to the region of the ligament of Treitz should be measured with a piece of tape which is kept for frequent use while preparing the jejunum. The marked tape is laid along the line of the vascular arcade of the mesentery to get an approximate idea of the length of jejunum required. The peritoneum of both sides of the mesentery of this length of jejunum is then totally removed from the root of the mesentery outward to the edge of the jejunum. This is facilitated and shock is reduced by injecting 0.5 per cent Novocain subperitoneally on both sides of the mesentery. Great care is taken to avoid even minor hemorrhage, lest an important anastomosing vessel might have to be tied. All nerve fibers running in the mesentery are divided, for they greatly limit the eventual straightening of the length of jejunum. The branches of the superior mesenteric artery and their accompanying veins are then individually ligated, close to the main artery, beginning with the first branch and proceeding distally until the required length of jejunum has been obtained. We have not had to divide more than four branches.

The jejunum is then divided about two inches distal to the ligament of Treitz, leaving just sufficient beyond the ligament to allow an end-to-side anastomosis to be made later. A clamp is left on the proximal end, but the distal end is tied and transfixed with heavy silk and the ligature left about eighteen inches long, so that subsequently it can

be passed up through the anterior mediastinum. The portion of jejunum to be transposed is now straightened out and any additional nerves or other tissue limiting this procedure are divided. We have found it possible to obtain greater length by ensuring that all peritoneum has been completely removed right up to the bowel. If secondary branches of the divided arteries limit the straightening of the jejunum, they may be divided if it is demonstrated that additional adequate anastomoses exist between them (Fig. 4). Veins must not be ligated any more freely than arteries, for venous drainage is just as important as arterial supply.

Having prepared the jejunum for its transposition, considerable time can be saved by continuing the operation as two surgical teams. One team completes the abdominal part of the procedure, while the other team exposes the esophagus in the neck and does the esophagojejunostomy.

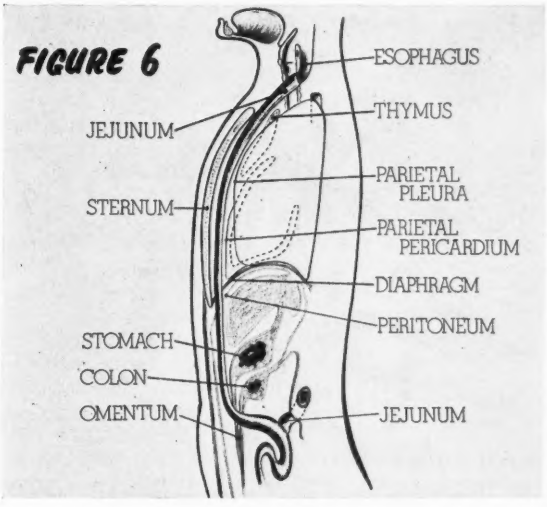


Fig. 6.—Sagittal section illustrating location of jejunal transplant in anterior mediastinum.

From the abdomen, the anterior mediastinum is entered (Fig. 6) through the anteromedial gap of the diaphragm, by blunt dissection upward and to either side, close to the deep surface of the xiphoid process and costal cartilages. If the xiphoid process is long, it can be removed. The diaphragm is then separated from the costal margin by blunt dissection for two inches on both sides of the midline. This allows the surgeon's entire hand to enter the anterior mediastinum. Blunt dissection with the fingers is gently carried upward anterior to the pericardial sac, carefully pushing both pleural reflections laterally. No troublesome hemorrhage has been encountered during this procedure. Gradually the hand is worked upward until it meets the fingers of the other surgeon working downward from the neck.

The cervical incision is made along the anterior border of the left sternomastoid muscle well down over the episternal notch. The left side is chosen as the lower cervical esophagus deviates slightly to the left. The deep fascia is then opened down into the suprasternal space, the left strap muscles divided, and the esophagus approached between the trachea and the carotid sheath (Fig. 7). The recurrent laryngeal nerve is then identified and protected and the cervical esophagus freed from the trachea and cervical spine and drawn up into the wound. The esophagus is clamped with slotted forceps below,

narrow forceps above, divided, and the stumps carbolized. The distal stump is then sutured with continuous chromic catgut through the slotted forceps and invaginated with an additional layer of interrupted black silk.

The anterior mediastinum is then tunnelled with the fingers from above keeping close to the sternum and separating the inner part of the insertion of the sternothyroid muscle. Space should be made to admit three fingers.

The long ends of the heavy silk tie on the proximal end of the jejunum to be transposed are now passed up through the anterior mediastinum and the jejunum very carefully drawn upward. Great care is taken not actually to pull on the narrow mesentery of the bowel for fear of injuring an artery or vein. The jejunum is pushed up through the mediastinum rather than pulled, and the silk ligature used as a guiding string. An attempt is made to lay the jejunum on its side and to avoid kinks, for the length of the bowel is always greater than the length of the mesentery.

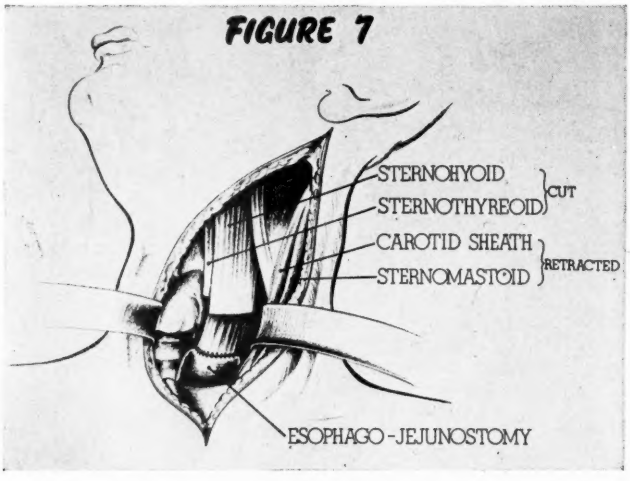


Fig. 7.—Jejunum anastomosed to esophagus in the neck.

Excess length of jejunum is excised and the upper esophagus divided above the narrow clamp. The esophagus and jejunum are then anastomosed meticulously with two layers of interrupted invaginating sutures placed 3 mm. apart. For the inner layer, 0 chromic catgut is used through all layers of the gut and securing a good bite of the esophageal mucosa. The outer layer is of 0 black silk and so placed as to invaginate the esophagus into the jejunum. The wound is then lightly powdered with sulfathiazole-penicillin mixture and closed with a Penrose drain.

Meanwhile an end-to-side anastomosis has been made between the short stump of jejunum left beyond the ligament of Treitz and the side of a convenient portion of jejunum.

A gastrostomy is made, using a mushroom catheter and bringing the catheter out 2 or 3 inches lateral to the paramedian incision.

The diaphragm is not sutured back to the costal margin and the peritoneum is very loosely closed around the jejunum as the latter enters the anterior mediastinum. The rest of the wound is closed in the usual manner.

The patient is fed intravenously for three days after operation, when gastrostomy feedings are commenced. Gastric suction is applied immediately for distention. Clear fluids are allowed by mouth after the second week and soft foods and solids after the third week.

CASE REPORTS

CASE 1.—Mrs. E. D., aged 50 years. On Sept. 23, 1948, thoracotomy revealed a carcinoma involving the lower esophagus and most of the stomach and infiltrating the pericardium, lung, and aorta.

Three weeks later an esophagojejunostomy was performed through the anterior mediastinum. An antiperistaltic tube was prepared by the method of Rienhoff and was a good color until drawn into the neck, where the end was blue and viability appeared doubtful. It was clear that excess tension had been applied, injuring the vessels of the arcade. However, after a few minutes the color improved slightly and the anastomosis was completed. At the end of the operation, the patient's blood pressure was 136/80 and pulse 100. Two days later she complained of a sense of oppression beneath the lower sternum, the pulse accelerated, and she died on the following day of gangrene of the jejunal tube.

CASE 2.—Mrs. S. B., aged 70 years. Patient admitted to the Vancouver General Hospital on Oct. 28, 1948, with a carcinoma of the lower third of the esophagus and secondary deposits in the cervical lymph nodes. She was very emaciated, weighing only 85 pounds.

Twelve days later a cervical esophagojejunostomy was performed. Secondary tumors were found in the celiac lymph nodes, liver, and mesentery. As in the first case, an antiperistaltic tube of jejunum was used and a gastrostomy was not done. Condition of patient at end of operation was good with blood pressure 130/90 and pulse 80. Two days later the neck incision was clean and so the Penrose drain was removed. Sips of water were commenced by mouth, but the patient vomited bile-stained fluid and continued to do so without letup. Eight days after operation both the neck and abdominal incisions were clean and sutures were removed, but the persistent vomiting commenced to produce feculent fluid. Two days later this fluid first discharged from the neck incision.

In the fourth postoperative week the abdomen was reopened. The jejunal tube was hypertrophied, and contracted firmly when touched. The jejunojejunostomy was well healed and widely patent, yet the bowel distal to it was absolutely collapsed. Obviously the stoma was not functioning yet no adequate reason could be seen. A second side-to-side jejunostomy was performed below the first and a catheter jejunostomy established for feeding lower down. The feculent vomiting continued and the patient died a few days later.

At autopsy the jejunal tube was viable. The fistula in the neck from the esophagojejunostomy was 5 mm. in diameter; otherwise the anastomosis was intact.

At the time it was felt that the vomiting and incomplete wound healing were the result of widespread tumor metastases. Unfortunately, the association of the vomiting with the aniperistaltic type of jejunal tube was not yet fully appreciated.

CASE 3.—Mr. M. J. McL., aged 69 years. March 1, 1949, patient was admitted to Shaughnessy Veterans Hospital and found to have a squamous carcinoma involving the lower 9 cm. of the esophagus. It was realized that a supra-aortic esophageal section would be required. A preliminary esophagojejunostomy was planned to be followed by a second stage for complete removal of the thoracic esophagus and the cardia of the stomach.

A cervical esophagojejunostomy was performed with a jejunal tube through the anterior mediastinum. An antiperistaltic tube was again used but in addition a catheter was inserted into the jejunum for feeding. At end of operation blood pressure was 123/70 and pulse 92.

Four days after operation his condition was good and clear fluids were commenced by mouth. The next day he began to complain of nausea and increasing pain and pressure in the upper abdomen. Various measures failed to give relief and he became increasingly distended and short of breath, and died on the eighth postoperative day.

Autopsy showed acute dilatation of the stomach, duodenum, and transplanted jejunum. There was no gas in the stomach which accounted for the failure to make a diagnosis. The jejunojejunostomy was patent and well healed but apparently not function-

ing. The neck wound was well healed but the esophagojejunostomy although intact was not safely healed, for although the esophageal muscle and jejunal peritoneum had adhered, the mucosal layers were not continuous (Fig. 8).

We were then forced to the conclusion that an isoperistaltic tube was essential to a well functioning jejunojejunostomy. In addition a gastrostomy is necessary for prolonged feeding to permit secure healing of the esophagojejunostomy.

CASE 4.—Mr. C. T., aged 43 years. Patient was admitted to the Vancouver General Hospital on April 14, 1949, having swallowed lye five weeks before. Barium swallow showed the esophagus occluded beneath the manubrium (Fig. 9, *A*). While being prepared for esophageal reconstruction an emergency operation was necessary for perforation of the stomach which was found to be shrunken and indurated as well as ulcerated. At the same time a catheter gastrostomy was made for feeding.

Ten days later a cervical esophagojejunostomy was constructed through the anterior mediastinum using an isoperistaltic tube and other measures as described above in our present technique (Fig. 9, *B*).

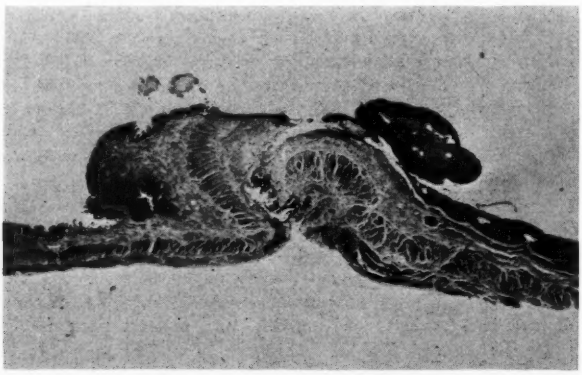


Fig. 8.—Longitudinal section at site of esophagojejunostomy showing extent of healing eight days after operation. The esophageal muscle and jejunal serosa have adhered but the gap in the mucosa remains.

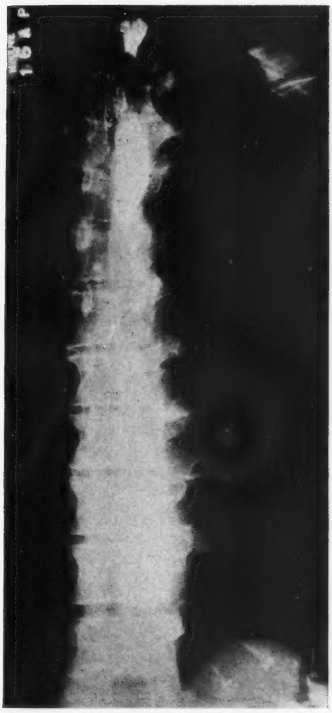
A month later he was able to swallow fluids only. At esophagoscopy the invaginated edges of the esophagus at the anastomosis were found adherent to each other. They were readily freed with bougies and separated widely leaving a large lumen. A barium swallow five weeks after operation showed jejunal transplant in the anterior mediastinum functioning well (Fig. 9, *C*). Subsequently he has been on a full diet with only occasional difficulty in swallowing leafy vegetables.

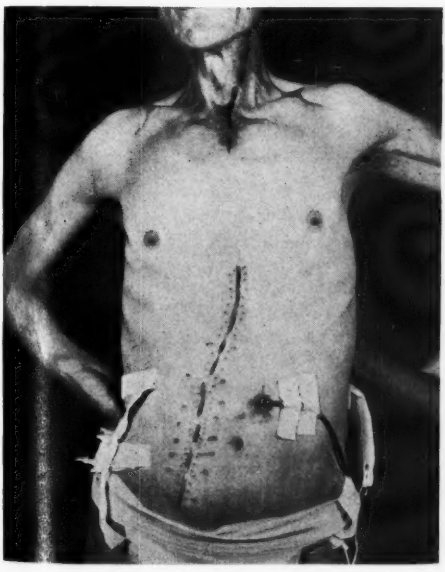
In June he developed signs of a penetrating gastric ulcer and on June 13 a total gastrectomy was performed. The lower stump of the esophagus was sutured and invaginated. The excised stomach was extensively scarred and ulcerated from lye burns especially along the lesser curvature.

At present he is working full time on the farm, eats heartily, and weighs 180 pounds. The isolated esophagus has not caused trouble. It seems likely that the mucosa has been entirely destroyed.

CASE 5.—Mr. G. S., aged 69 years. Patient was admitted to the Vancouver General Hospital on July 3, 1949, with a carcinoma of the esophagus, ulcerating into the left main bronchus, and occluding the esophagus (Fig. 10, *A*). Patient was emaciated, weighed only 105 pounds, his heart was fibrillating, and there were râles at the base of both lungs.

A.





B.

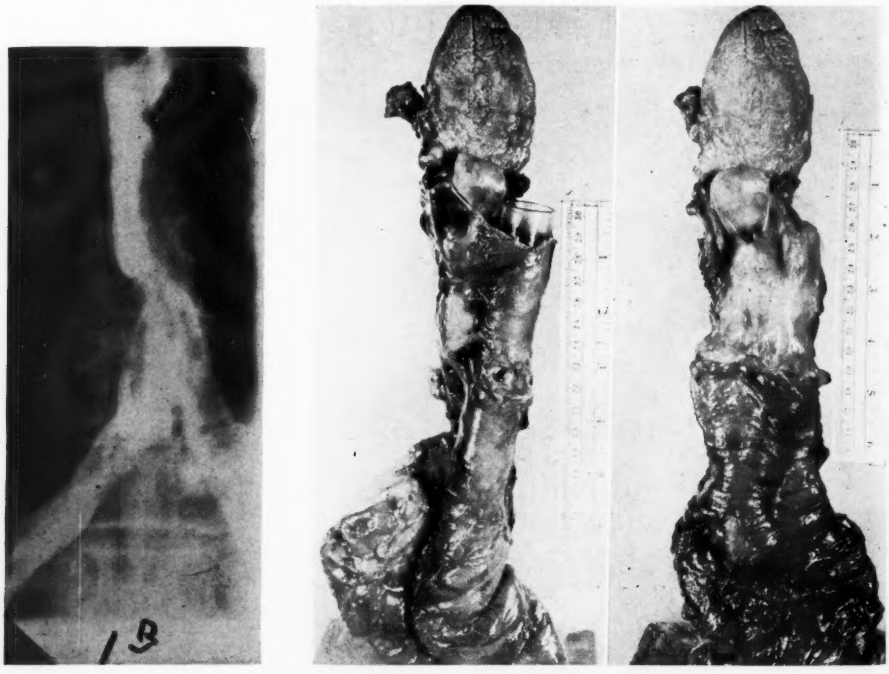
C.

Fig. 9 (Case 4).—*A*, Preoperative barium swallow. *B*, Three weeks postoperative, showing neck and abdominal incision and gastrostomy tube. *C*, Five weeks postoperative, barium swallow showing jejunal transplant in anterior mediastinum.

On July 28, 1949, a palliative esophagojejunostomy was performed as described in our present technique. Subsequently he was able to take small amounts of fluid and soft food with some satisfaction.

Two months after operation he died of bronchopneumonia, apparently from regurgitation of stomach contents through the esophagobronchial fistula.

At autopsy the esophagojejunal anastomosis was completely healed with a large lumen (Fig. 10, *B* and *C*).



A. *B.* *C.*

Fig. 10.—(Case 5) *A*, Barium swallow showing esophagobronchial fistula from ulcerating carcinoma of esophagus. *B*, Autopsy specimen showing size of lumen at jejunoesophagostomy. *C*, Autopsy specimen showing level and quality of anastomosis.

CASE 6.—Mr. L. T., aged 36 years. On April 16, 1948, the left lung was removed for Hodgkin's granuloma involving the left main bronchus and forming a tumor the size of a grapefruit in the left hilus. Eight months later the tumor recurred in the lower trachea and ulcerated into the esophagus and left chest (Fig. 11, *A*). An intercostal catheter was inserted and x-ray therapy repeated. The fistula persisted and swallowing of food or saliva initiated a most distressing cough.

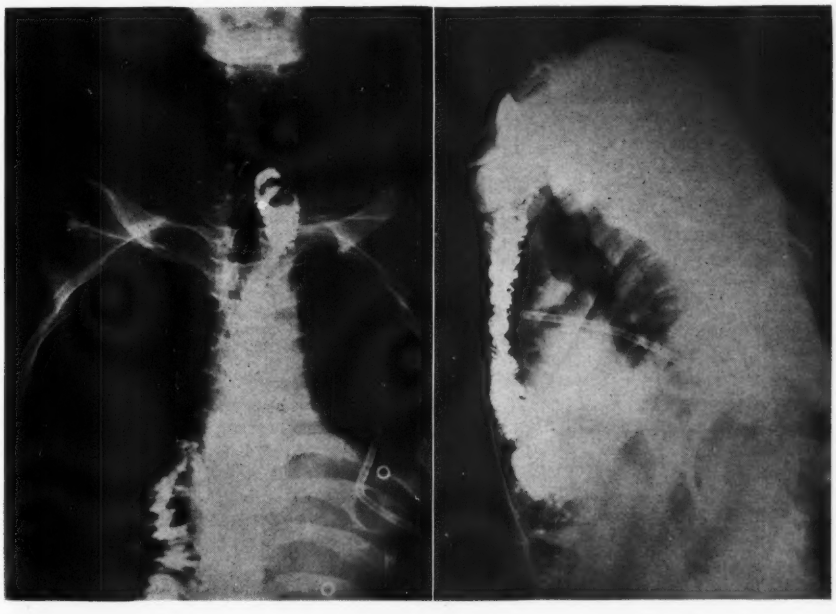
On Oct. 25, 1949, an esophagojejunostomy was performed as described previously (Fig. 11, *B*). At the conclusion of the operation his condition was good with blood pressure 110/70 and pulse 72.

Five weeks after operation he was taking a full diet readily and a barium swallow showed the reconstructed esophagus functioning freely (Fig. 11, *C*). He remains fairly well and has gained twenty pounds (Fig. 11, *E*).

A.

B.

Fig. 11 *A* and *B*.—(Case 6) *A*, Barium swallow showing tracheoesophagopleural fistula. *B*, Patient two weeks after esophagojejunostomy.



C.

Fig. 11 *C*.—(Case 6) *C*, Five weeks postoperatively. Barium swallow showing transplanted jejunum beneath sternum.

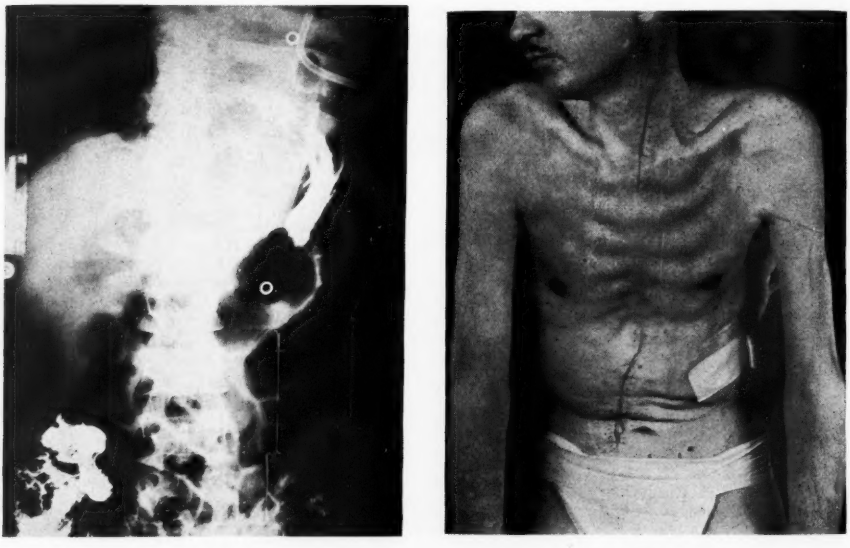
CONCLUSIONS

1. Reconstruction of the entire esophagus in one stage is possible with a minimum of shock.

2. A suitable tunnel can be formed through the anterior mediastinum for the transplanted jejunum. By this route a shorter loop of jejunum, with an adequate blood supply may be used. No symptoms of cardiac embarrassment have been noted beyond a feeling of fullness in the epigastrium after large meals.

3. An isoperistaltic tube of jejunum is essential.

4. A gastrostomy for decompression and feeding during convalescence is essential.



D. *E.*

Fig. 11 *D* and *E*.—(Case 6) *D*, Barium in stomach six hours after barium swallow through esophagojejunostomy. *E*, Patient four months after esophagojejunostomy.

5. The absence of a serous coat on the esophagus makes healing of the esophagojejunostomy slow and insecure.

6. Oral feedings should be long delayed.

7. Postoperative pain is minimal and does not interfere with the cough reflex.

8. The scarred esophagus following lye burns has not been removed and so far, has not caused trouble.

9. This procedure is most applicable to lye strictures extending into the upper esophagus. It would also be useful for the following:

A. As a first stage procedure for carcinoma of upper thoracic esophagus. Wide resection of the esophagus with the regional lymph nodes would then be possible.

B. As a palliative for inoperable carcinoma of the esophagus.
C. For carcinoma involving the stomach and lower esophagus requiring a complete gastrectomy.
D. For congenital atresias not treated by primary anastomosis.

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DISCUSSION OF "CONGENITAL ATRESIA OF THE ESOPHAGUS WITH TRACHEOESOPHAGEAL FISTULA"* AND "CONGENITAL ATRESIA OF THE ESOPHAGUS: ANTETHORACIC PLACEMENT OF THE STOMACH FOLLOWED BY INTRATHORACIC TRANSPLANTATION"† BY DR. WILLIS J. POTTS; AND "RECONSTRUCTION OF ESOPHAGUS" BY DR. ROSS ROBERTSON AND DR. T. R. SARJEANT

DR. CAMERON HAIGHT, Ann Arbor.—It is always a pleasure to hear a paper by Dr. Potts, because he not only presents a new thought but also presents it in a very stimulating and entertaining manner. He has told you of the difficulties one is likely to encounter in infants with congenital atresia of the esophagus with or without tracheoesophageal fistula and I do not believe he has overstated them. The desiderata that one likes to see in surgery, that is, 100 per cent recoveries, is, I feel, impossible in these cases for the reason that the associated anomalies, the poor condition of some of these patients, and the other factors over which we have little control, will mitigate against obtaining perfect results.

(Slide.) This slide depicts the results of the 93 cases seen in our clinic in the interval between 1939 and the first of this year. Eighty-six of the patients were operated upon, with 41 operative recoveries and 3 late deaths from conditions apart from operation. You will note on the slide the date (1944) when penicillin was first used and the date

*See p. 671 for article by Potts.
†See p. 681 for article by Potts.

(1947) when streptomycin was begun. Although penicillin has been distinctly helpful, it has not been the panacea we thought it would be, because of the frequent presence of penicillin-resistant organisms. Our results have been distinctly better since streptomycin has been employed in conjunction with penicillin. In the 42 cases treated during the last three years, there have been 29 recoveries and 13 deaths following operation.

In order to reduce the mortality further, it seems to me that it is necessary for us to modify our previous philosophy regarding the multiple-stage plan which we formerly avoided. It used to be our endeavor to obtain esophageal continuity by direct anastomosis even at very long odds. If one takes such long chances because of too great separation of the segments, a certain number of the anastomoses will break down and the mortality in this group will be considerable. In the last year we have started the multiple-stage plan in several cases when an esophageal anastomosis did not seem advisable. It is planned to obtain intrathoracic continuity at a later date by esophagogastrostomy or by esophagojejunostomy, as described previously by Dr. A. W. Harrison or as described today by Drs. Robertson and Sarjeant. In our cases, three primary esophagogastrostomies have been done by other members of our staff, one of them by Dr. C. F. Storey being successful. By and large, however, this operation as a primary procedure in the infants without air in the stomach carries a high risk and, therefore, I feel that the safer plan is to tide such infants along with gastrostomy and cervical esophagostomy until such time as they can more safely withstand esophagogastrostomy or esophagojejunostomy.

The operative approach as described today by Dr. Potts, and earlier by Dr. I. A. Bigger, has much to be said in its favor, although I have not used it as yet, inasmuch as the right extrapleural approach with the resection of short lengths of three ribs posteriorly has given adequate exposure.

DR. LOGAN LEVEN, St. Paul, Minn.—It was a pleasure to hear Dr. Potts' and Dr. Robertson's papers. We have had some experience with tracheoesophageal fistula at the University Hospital in Minnesota. Since 1939 we have seen 77 cases, and in only 5 the operation was not carried out either because the patient died in the receiving room or the parents refused permission for surgery. In our earlier experience we performed a gastrostomy on 5 patients. This probably was worse than no operation at all, since it added to the regurgitation through the tracheobronchial fistula into the lungs. In our first cases we performed the so-called multiple-stage operation, doing 20 such cases. I believe that the multiple-stage operation will always have a place in the management of some of these cases. In the simple atresias without tracheoesophageal fistula, the distance between the segments of esophagus is usually so great that a primary anastomosis cannot be carried out, and the risk of bringing the stomach into the chest as a primary procedure is exceedingly great at this age. Accordingly, I believe the multiple-stage operation would reduce the mortality in such cases.

Of the 20 in which we performed a multiple-stage operation, 11 have survived. I am sure at the present time this mortality would be improved considerably. Of the 11 surviving, six have had reconstructions, five by using an antethoracic jejunal tube and one by an intrathoracic operation, bringing the stomach into the cervical region and performing a primary anastomosis. Dr. Richard Varco has performed these reconstructions with no mortality. The use of the jejunal tube antethoracically may have considerable merit. The stomach is left in the normal position with intact vagus nerves. The criticism of the undesirable cosmetic appearance could be corrected by division of the ribs and replacing the jejunal tube within the chest after it has been successfully reconstructed antethoracically, as Dr. Potts did with the stomach in his case. The risk of bringing the jejunal tube subthoracically would be considerably less than bringing it up through the thorax as a primary procedure. In none of our cases have we lost any of our jejunal tubes. However, an anastomosis of the jejunal tube to the cervical esophagus was not carried out as a primary procedure but performed after an interval of several weeks.

We have carried out primary anastomosis in 47 cases and have 30 that are living. The results in the last few years have been considerably better than was our initial experience. In the last two years we have performed 19 primary anastomoses, with 15 patients living

and well. It is of considerable interest that these 19 cases have been done by seven different surgeons of the Surgery Department of the University of Minnesota Hospital.

We have used an incision in the right chest, removing the third or fourth rib in its entirety in all our cases since the first successful case in 1939, when we did a multiple-stage procedure, ligating the tracheoesophageal fistula extrapleurally. I feel that this incision gives very adequate exposure and agree with Dr. Potts that a satisfactory chest wall is the end result.

I should like to emphasize the limitation of fluids and, especially, the limitation of saline in the postoperative period. In most of our cases we perform a gastrostomy the day following primary anastomosis of the esophagus. In 10 cases we did not perform a gastrostomy, but I feel that it is a safety precaution which facilitates the feeding problem in these cases in the postoperative period.

In all our cases we have used cyclopropane, and in the last several years have used intratracheal anesthesia. In none of the cases have we had difficulty incident to the anesthesia.

DR. GEORGE H. HUMPHREYS, NEW YORK.—I am sorry I cannot give detailed statistics, but at the Babies' Hospital in New York we have had experience with approximately 50 of these procedures over the past ten years. The philosophy back of the procedure is interesting. I seem to have gone through exactly the reverse of Dr. Haight's change in philosophy. We started out by doing mostly multiple-stage operations, bringing the esophagus up into the neck and closing the fistula. We now try to anastomose them, even at long odds. I think that change in philosophy has been due to the difficulties in doing the secondary repair in these cases.

We do gastrostomies in all our patients. As Dr. Leven said, we feel that it is not always necessary when the repairs are satisfactory and the children can swallow probably very promptly. We know that is the case because the amount of mucus which is aspirated will vary with the patency of the anastomosis, and when the child does not have to be aspirated frequently the anastomosis is working well. However, we do feel it is safer not to feed early except by gastrostomy, and the dangers of parenteral over-hydration have already been emphasized.

We do not drain. We have drained in the past and have regretted it; we think sometimes drainage predisposes to leakage. Some leakage has occurred and has had to be drained secondarily, but if an anastomosis can be made satisfactorily and gastrostomy is done early, I think there will be very few leaks.

I would like to point out one fact which we have found out to our cost, which is in contrast to what Dr. Potts mentioned. That is, that absence of air in the stomach and intestines does not mean absence of a fistula. Some of these fistulas may be very small, the child may be very weak and does not have a strong enough cry or cough to force air through the fistula. In such a case there would be no air, especially in the first few days of life, in the intestinal tract, yet a fistula may be present.

Our mortality is high, approximately 30 to 35 per cent. Technical considerations play a part, but the prime factors are those emphasized by Dr. Potts, prematurity, failure to make the diagnosis early, and aspiration pneumonia.

DR. HARRIS B. SHUMACKER, JR., Indianapolis.—I should like to comment briefly about one aspect of the problem of utilizing the jejunum; namely, that of its blood supply. I think we all are in agreement that there are many cases in which the jejunum seems to be the ideal structure for use in esophageal reconstruction. Since the days when Roux first proposed the use of this organ, however, the one feature which has plagued operators has been the inadequacy of the blood supply of the freed and mobilized limb. It seemed to me that perhaps experiences with peripheral circulatory disorders might offer a suggestive lead. We know that the gradual occlusion of a major arterial stem is often tolerated without loss of tissue when the sudden occlusion of the same artery would ordinarily produce massive gangrene. Following this lead, Dr. Battersby and I have recently completed some animal experiments and one human application which I believe will go a long way toward solving this problem. We have found that loops of jejunum of a given length when freed of their arterial blood supply in one stage inevitably lead to gangrene and rupture, but maintain adequate circula-

tion if the division of the arterial supply is staged; first, dividing every other artery and, at a later date, dividing the intervening ones. If we divided in one stage the blood vessels to loops of the jejunum of the dog ranging from 25 to 40 cm. in length, gangrene and rupture inevitably took place. In contrast, if we divided every other vessel and, a week or ten days later, divided the intervening ones, we had segments as long as 55 centimeters maintain good circulation. Indeed, by using three stages in one animal we obtained a Roux-Y limb, which was almost 100 cm. long and which was successfully transplanted into the neck.

I think the results are well illustrated in two lantern slides which I should like to show. In the rat, if one ligates four consecutive jejunal arteries, inevitably the intestine immediately becomes cyanotic, as is illustrated in the first slide, and it subsequently becomes gangrenous and ruptures. As is shown in the next slide, a Roux-Y can be constructed and six consecutive vessels ligated in two stages with maintenance of good circulation.

In one human being with a total esophageal stricture from lye injury, this principle has worked extraordinarily well. A Roux-Y jejunal limb was made and some of the vessels divided; ten days later the remaining vessels were divided and the jejunum transplanted; a week later the anastomosis was performed.

DR. OSLER A. ABBOTT, Atlanta.—I should like to comment on two aspects of the tracheoesophageal problem. We are becoming increasingly convinced that Lipiodol should not be used in the study of these patients because of the frequency with which it may spill over into the tracheobronchial tree and carry with it a consideable amount of inspissated material from the upper esophageal segment, which is a potential serious danger. We are trying to emphasize to pediatricians in our area to make the diagnosis by insertion of a catheter alone, and if Lipiodol is insisted upon, that a preliminary clean-out of the upper esophageal stump be done. We have had several x-ray pictures sent to us showing Lipiodol spilling over from a blocked upper stump into the tracheobronchial tree.

I should like to mention that we have had experience with two patients of the same type as Dr. Haight presented, without atresia, but with tracheoesophageal fistula of congenital type. One of these was discovered, and operated upon successfully, within the first six weeks of life. We also feel that one of the greatest help which one of our staff obtained recently from a visit to Dr. Haight's clinic, is the importance of keeping the child on the side to be operated upon, for twenty-four hours prior to operation, so that all the inflammatory and other trouble in the lung may be confined to that area. This has been of tremendous value to us and, I think, deserves emphasis.

DR. CONRAD LAM, Detroit.—I thought there might be some interest in a progress report on the two boys whose pictures I showed two years ago in Quebec. If you recall, the continuity of the esophagus in each was obtained by the somewhat outmoded method of skin tube construction. The oldest child I have not seen professionally, but have seen socially a few times; he is now in second grade in school and apparently is suffering no psychologically bad effects from his antethoracic esophagus. I should like to ask Dr. Potts if he thinks this esophagus could be transplanted to an intrathoracic position by his method.

The second child, I must report, is still alive and healthy, but about six months ago he had what appeared to be acute follicular tonsillitis, and the inflammation spread down into the chest about the skin tube. I had to insert a tube for feeding for a few days. This little opening has persisted as a sinus and shows little tendency to close, although the only thing that is necessary for this boy to eat three meals a day is for the mother to put a small piece of adhesive tape over it.

DR. JULIAN JOHNSON, Philadelphia.—There is one point I would like to mention. I am sure that a good many people over the country are doing these tracheoesophageal fistulas by the transpleural approach. We have been using it for the last year or eighteen months at Children's Hospital in Philadelphia. Making a fourth intercostal incision, we go transpleural and the operation can be done much more quickly, usually being completed in an hour and fifteen minutes to an hour and a half. It seems to us that the only risk is that there might be a leak at the point of anastomosis, resulting in generalized empyema. We have had one leak of this type; it has not produced a generalized empyema, however. By the time

the leak occurred there was apparently sufficient sticking of the lung around that area so that the tube which had been placed in the manner described by Dr. Potts took care of the leak satisfactorily. We think the operation can be done much more quickly; we think the children do not have as much respiratory difficulty postoperatively and, all in all, in our short experience we have been favorably impressed.

DR. R. H. SWEET, Boston.—There are one or two points on which I would like to comment. One is about the use of the stomach to restore continuity in these multiple-stage cases of which Dr. Potts and others have spoken. It seems to me that his experience has quite amply demonstrated that the better place for the stomach is in the chest rather than in the subcutaneous area. This, of course, can be done as a primary procedure and, in my experience, has worked well. I would like, however, to lend emphasis to some of the comments this morning, that it is unwise to do that operation in an infant, because of the disparity in size between the enormous fluid and gas filled stomach and the volume of the thorax. In my hands, at least, it has not been a successful procedure. I much prefer to delay and use the stomach later.

The other comment has to do with the use of the jejunum. The essayists and commentators this morning have brought up some of the difficulties. Dr. Robertson referred to the experience I had with a patient presented before this Association in the discussion of Dr. Harrison's report, which I believe lends weight to the wisdom of Dr. Harrison's point of view that the lower end of the jejunal segment should be implanted into the stomach. That patient behaved very much like a person who had a jejunostomy performed for feeding, and we were unable to maintain a satisfactory degree of nutrition. She lost weight at home and the only way we could keep her in good condition was to keep her under supervision in the hospital. At a later date I reoperated upon her and transected the loop of jejunum above the jejunojejunostomy, implanting the proximal end of the transected portion into the fundus of the stomach. Since then she has maintained a satisfactory nutritional state.

DR. POTTS (closing).—Many interesting and valuable points have been brought out in the discussion. Dr. Haight asked about the virtues of resection of a rib instead of the subperiosteal resection of three ribs. We believe that we get better exposure in that way, because the ribs of an infant are extremely flexible. When one rib is removed a small mastoid retractor will easily spread the ribs widely. In closing the wound these very flexible ribs can be brought together; if the fourth is resected the third can be sewed to the fifth and thus furnish a solid chest wall against which the child can breathe.

I was glad to hear the tendency toward intrathoracic transplantation of the jejunum as an esophagus. I think Robertson's suggestion of putting it behind the sternum is an excellent one. After all, these children put themselves unwittingly into our hands and are entirely helpless in what we do. The parents cannot evaluate our advice. I do not believe that a girl should have an antethoracic transplantation of anything—jejunum or anything else. It is going to be a very peculiar deformity at the age of 16 years. I question whether it should be done on a boy. After all, to live nobly, as Dr. Eloesser has said, is such an extremely important thing. The psychologic effect of having such an abnormal structure on the front of the chest, when the youngster goes to high school and college, I believe will be disturbing. I am sure all efforts should be made toward intrathoracic construction of esophageal tubes.

DR. ROBERTSON (closing).—I wish to thank Dr. Haight particularly for his indication that he may be tempted to use a jejunal transplant in the anterior mediastinum. Regarding the problem of nutrition in these patients, as brought out by Dr. Sweet, we have anticipated difficulty in that regard, but so far the two patients that have done well have gained a great deal of weight; one man has gained about sixty pounds and the other man, with Hodgkins' disease, has gained twenty pounds, and we feel we will reserve the transplant into the stomach for patients who fail to gain weight.

Postoperative x-ray films on these patients show some retrograde filling of the stomach. I believe Dr. Harrison also had that experience.

USE OF THE BURNS VALVE AS A SIMPLE RESPIRATOR FOR INTRATHORACIC SURGERY IN THE DOG

JOSEPH T. DAMERON, M.D.* (BY INVITATION), AND DAVID G. GREENE, M.D.†
BUFFALO, N. Y.

THE efforts of members of the surgical staff in this laboratory were handicapped by the lack of a simple respirator which did not produce anoxia and a progressive increase in carbon dioxide content of the blood when attempting various intrathoracic operations in the dog.

Use of the "windshield wiper" respirator[1] used in many laboratories resulted in the death on the table of many of our animals from anoxia and ventricular fibrillation. This machine operates by giving a sudden blast of air into the trachea for inflating the lungs. The degree of deflation of the lungs depends upon the amount of leakage around the intratracheal tube. The intratracheal pressure during inspiration and expiration is uncertain. Sustained intratracheal pressure during expiration greater than atmospheric pressure impedes the venous return to the heart and consequently cardiac output. This laboratory has had no personal experience with either the Crafoord[2] or Mautz[3, 4] machines. Both are expensive and require a skilled attendant. Gibbon's data[5] suggest these two machines do not always overcome a tendency for a rise in the arterial CO_2 tension. Gunkler and Mahoney[6] have recently described a complicated respirator for intrathoracic surgery without giving either blood gas values or the intratracheal pressure during inspiration and expiration.

The Burns valve[7] was developed during the last war for the Air Force as a respirator. The mechanics of the valve may be found in Burns's original paper. The purpose of this paper is to report: (1) the adaptation of this valve for anesthesia during intrathoracic surgery in the dog and cat; and (2) to present data on the arterial blood gas values in the dog, when operating the valve with either compressed air or 100 per cent oxygen.

METHODS

A. *Description.*—Over 150 dogs weighing between 12 and 75 pounds have undergone prolonged intrathoracic operations using the method to be described. This device has also been adapted to the cat. The preoperative medication in the dog consists of morphine sulphate 0.015 Gm. and atropine sulfate 0.0004 Gm. given intramuscularly one hour pre-

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operatively. The animal is anesthetized lightly with either pentothal or pentobarbital by the intravenous route. A No. 36 Colby hemostatic catheter, which may be found on most urological services, is inserted into the trachea and the bag inflated for a tight seal. We have found this catheter to make an excellent intratracheal tube. It has a durable bag which is not easily punctured. A No. 18 Foley catheter with the tip cut off is easily adaptable as an intratracheal tube for the cat. An ether bottle is interposed between the Burns valve and the intratracheal tube. If the line pressure is set to cut off at a pressure of 12 to 14 cm. of water or less, the lungs will rhythmically inflate 14 to 20 times per minute to the desired pressure, and supplementary ether may be given as necessary. The 11 animals in which blood gas analyses were made were completely anesthetized with pentobarbital, since slight traces of ether might invalidate the blood gas determinations. Fig. 1 demonstrates the apparatus as used.

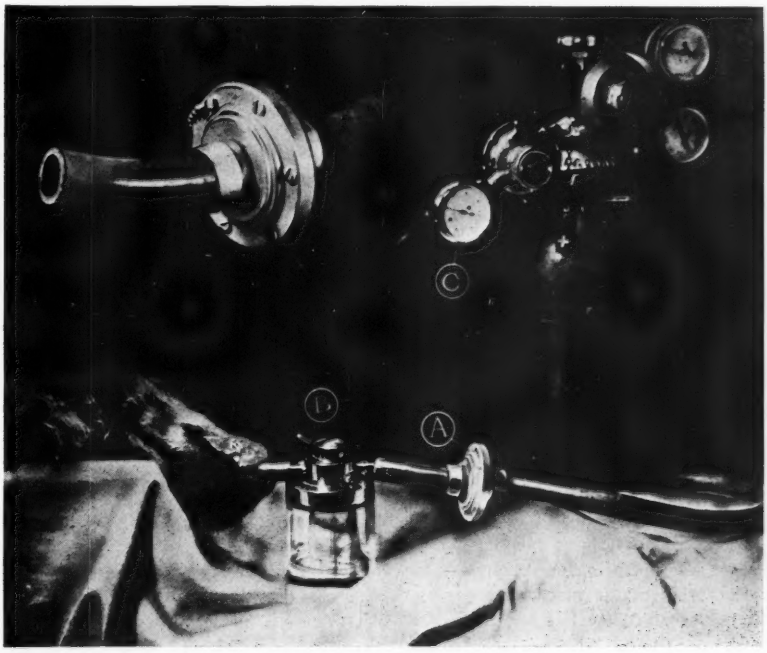

Fig. 1.—The apparatus as used: *A*, Burns valve. *B*, Ether bottle with regulator. *C*, Pressure gauge and regulator for maximum pressure.

B. *Blood Gas Analysis.*—Arterial blood was obtained from the femoral artery of 11 dogs. In the 7 dogs in which compressed air was used, the partial pressures of carbon dioxide and oyygen (pCO_2 and pO_2) were obtained by the bubble technique of Riley and associates.[8] In 4 dogs in which 100 per cent oxygen was used, arterial blood samples were analyzed for oxygen and carbon dioxide content, and oxygen capacity by the method of Van Slyke and Neill,[9] and the pH was measured with a glass electrode. From these figures the pCO_2 was calculated using the nomogram of Singer and Hastings[10] for human blood. In all 11 experiments, one sample of arterial blood was taken after the dog was premedicated with morphine and anesthetized with pentobarbital prior to opening the chest and connecting

the respirator. This is labeled control sample. Subsequent samples were obtained after the chest had been opened and the respirator functioning for one and one-half to three and one-half hours.

The intratracheal pressure in dog No. 208 was recorded graphically, using a Hathway blood pressure recording system, and is reproduced in Fig. 2.

RESULTS

The Burns valve has been used successfully in over 150 prolonged intrathoracic operations in the dog and in 6 cats. It has eliminated the need for an anesthetic attendant in the operating room. No dog has died on the operating room table while the valve was in use, a record which is in striking contrast to our experience with other methods.

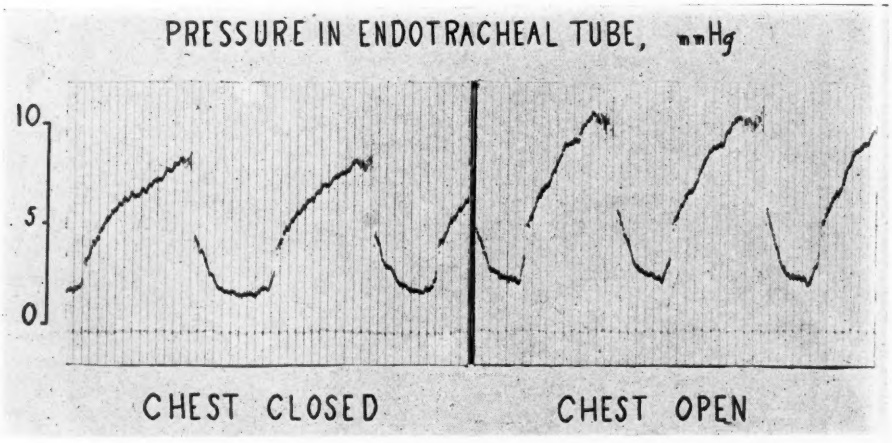

Fig. 2.—Variations in pressure throughout the respiratory cycle, measured in the intratracheal tube. Time lines indicate 0.1 second intervals. Dog 208.

The partial pressures of carbon dioxide and oxygen are recorded graphically in Fig. 3 and a detailed analysis of the blood is given in Table I. One will note that when the respirator was operating on compressed air, approximately 21 per cent oxygen, the carbon dioxide tension of the blood never rose above normal limits and in most instances there was a decrease. When the respirator was operated on 100 per cent oxygen, the tendency to blow off carbon dioxide excessively was not noted. In 2 dogs a high carbon dioxide tension was noted with the use of 100 per cent oxygen. Acidosis was noted after the use of the respirator and 100 per cent oxygen in all 4 dogs in which the pH of the arterial blood was measured. This had decreased in comparison with the control value in 2 dogs, in one it developed although the control value was normal and in one it remained essentially the same as the control value.

The shape of the intratracheal pressure curve, Fig. 2, shows a fairly gradual rise in pressure during inspiration, and a rapid drop at the beginning of expiration followed by a more gradual decline which never reaches atmospheric pressure. The inspiratory phase is longer than the expiratory phase of respiration. Although the peak inspiratory pressure is higher in the tracings taken with the chest open, the general shape shows little change. The increase in peak pressure is due to a slight change in line pressure between the two tracings.

TABLE I

CHEST CLOSED, NO RESPIRATOR, ANESTHETIZED (CONTROL SAMPLE)			CHEST OPEN, WITH RESPIRATOR			
			21% OXYGEN		100% OXYGEN	
DOG NUMBER	pCO_2 (MM. Hg)	pO_2 (MM. Hg)	pCO_2 (MM. Hg)	pO_2 (MM. Hg)	pCO_2 (MM. Hg)	pO_2 (MM. Hg)
111	52	51	15	105	–	–
171	43	93	25	111	–	–
173	40	45	38	61	–	–
135	50	80	45	71	–	–
162	46	78	37	66	–	–
164*	34	67	9	88	21	145+
187*	41	84	28	69	34	145+

					100% OXYGEN			
	CO_2 CONT. (VOLS. %)	pCO_2 (MM. Hg)	pH	O_2 SAT. %	CO_2 CONT. (VOLS. %)	pCO_2 (MM. Hg)	pH	O_2 SAT. %
201	46.6	64	7.16	82.4	40.3	46	7.26	100+
231	43.3	57	7.17	83.4	35.9	49	7.28	100+
207	43.4	36	7.43	81.8	43.8	65	7.15	100+
217	47.9	60	7.18	83.6	44.7	60	7.16	96.6

*Dogs maintained on respirator with compressed air (21 per cent O_2) for two hours and second sample then obtained. The third sample obtained after further maintenance on respirator for thirty minutes with 100 per cent O_2. Approximate tensions estimated by Riley technique.

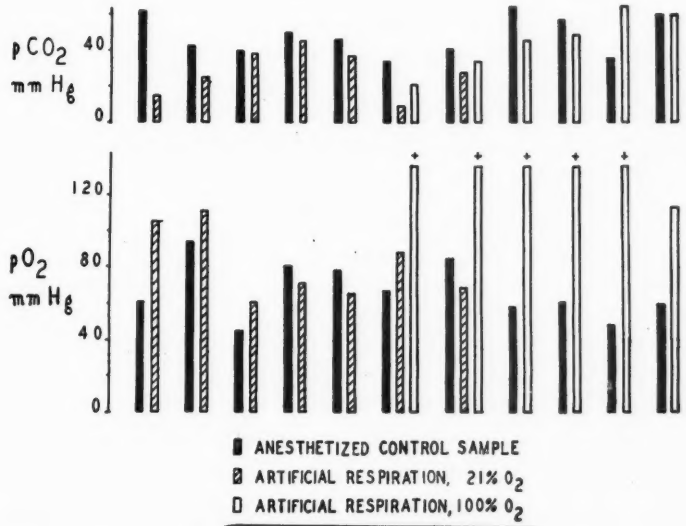

Fig. 3.—Blood gas results expressed in terms of carbon dioxide and oxygen tensions, ther measured directly or calculated. Plus marks indicate that oxygen tension was 145 m. Hg or greater. Normal values for carbon dioxide tension 36 to 48 mm. Hg, for oxygen nsion 80 to 100 mm. Hg. The graphic illustration was obtained on eleven animals, each oup of columns having been obtained on a separate animal.

DISCUSSION

The advantages of the Burns valve for anesthesia in intrathoracic surgery are several. It operates from a tank of oxygen or compressed air and requires no motors, gears, or switches which may be explosive hazards. The lungs may be expanded rhythmically without danger of exceeding a predetermined peak pressure. A trained individual is not needed to operate the device, and it is compact, simple, and inexpensive to operate.

The experiments using compressed air show that under these circumstances there is no tendency for carbon dioxide tension to increase, but that on the contrary it sometimes reaches low levels. In 3 dogs a satisfactory level of oxygen tension was maintained, but in 4 others there was slight or moderate hypoxia. This hypoxia has not been a problem practically, as it can easily be overcome by the use of 100 per cent oxygen as the respiratory gas. The tendency of the carbon dioxide tension to remain at or rise to an abnormal high level in 2 dogs on 100 per cent oxygen is an interesting contrast to the fall in carbon dioxide tension on compressed air.

When compressed air is used there is either a normal or diminished oxygen tension, and the stimulus of anoxia might be expected to play a part in preventing a further reduction in ventilation. When 100 per cent oxygen was used, the oxygen tension is uniformly above normal, and the only chemical stimulus to ventilation must be mediated by CO_2. If the threshold of the respiratory center for hypercapnia or acidosis be elevated, hypoventilation and CO_2 retention might easily result. The samples taken before artificial respiration was begun show that hypoventilation was sometimes present simply as the result of depression of the respiratory center. Morphine and pentobarbital are both known to depress the respiratory center.

It may be suggested that the respiratory center is not involved in artificial respiration. This is not completely true of the Burns valve which tends to follow the natural rhythm of respiration to a certain degree when respiratory movements are made. The high arterial oxygen tension then may result in hypoventilation through diminished stimulation of the respiratory center. An analogous mechanism presumably mediated through the respiratory center has been described by Baldwin and co-workers[11] in certain patients with chronic pulmonary emphysema who develop an increase in respiratory acidosis when oxygen is given. Furthermore, oxygen saturation of the arterial blood, as judged by the color in cut vessels and capillaries, is one of the chief criteria for regulating the artificial respirator during the operation. High oxygen concentrations in the inspired air would prevent cyanosis from indicating to the operator the need for more ventilation.

One further explanation of these findings is possible, so-called "oxygen poisoning."[12] Concentrations of oxygen greater than 70 per cent have been demonstrated to produce various lesions in the lung when administered over periods of time exceeding six hours, or when given at greater than atmospheric pressures. Two or three hours of pure oxygen at atmospheric pressure would not seem likely to produce lung damage.

Hyperventilation beyond what seems required by the respiratory demands of the dog and the color of the arterial blood might be useful to overcome hypercapnia. This possibility is distinctly limited with the particular type of valve involved, which has been shown by Motley and associates[13] to produce a fall in cardiac output which is proportional to the line pressure above a certain level. Fig. 2 shows that the pressure curve in the intratracheal tube in the anesthetized dog is similar to that described by Motley in man with the same apparatus.

Beecher and Murphy[14] have called attention to the importance of respiratory acidosis in thoracic surgery. They found that carbon dioxide retention was a more serious problem than oxygen unsaturation, and that it could not always be relieved by "assisted respiration." While Beecher has stressed that it is not enough to "get away with" an intrathoracic operation, it nevertheless is of significance that no dogs have been lost on the table when the artificial respirator was in use. The respiratory acidosis which must have been present in many cannot have been desirable, but from a practical standpoint it has not been a serious problem in prolonged experimental intrathoracic surgery.

Since submission of an abstract of this paper for today's meeting, Adelman, Berman, and Touroff[15] have reported the successful use of the Burns valve for respiration during pentothal-curare anesthesia in 16 patients. Touroff[16] in a recent personal communication stated that the valve has been used in 20 selected cases. He is satisfied with the results obtained. In our adaptation of the valve, ether has been successfully used to supplement the intravenous anesthetic. This flexibility in the choice of anesthetic agents may be desirable clinically.

SUMMARY

A simple method of rhythmically expanding the lungs during intrathoracic surgery has been described. The efficacy has been tested extensively in practice and by analysis of arterial oxygen and carbon dioxide tensions in eleven experiments.

Acknowledgments.—The authors wish to thank Mr. John B. Dunne and Mr. Lewis E. Fancourt of the Mine Safety Appliance Company, Pittsburgh, Pa., for the apparatus herein described, and Miss Mary Ann Grammer for technical assistance.

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DISCUSSION

DR. F. R. MAUTZ, Cleveland.—My interest in mechanically controlled breathing also originated in the surgical laboratory, and about twelve years ago we began to use mechanically controlled breathing in clinical thoracic surgery. We found some differences between the dog and man due, presumably, to such factors as relative size, vigor of the pulmonary reflexes, anatomic differences in the pleural membrane, and such pathologic processes as pleural thickening, adhesions, and pulmonary fibrosis, with emphysema sometimes present in man. As a rule these pathologic processes were not present in the dog.

In our earlier experiences these factors gave us some difficulty in taking over mechanical control of breathing without resorting to a depth of anesthesia that might be detrimental. However, following the introduction of curare, our clinical problem was greatly simplified, and at present a future seems assured for mechanically controlled breathing. We have now used mechanically controlled breathing in approximately 300 patients, controlling respiration from the time just before the pleura is opened until the pleura is closed, and we feel that patients can be kept in a better physiologic state for procedures of considerable duration by this method than by other methods of management. I have no quarrel with the anesthetist who wants to get his exercise by manual compression of the breathing bag, provided he will supply the necessary amount of pulmonary ventilation. I think in the future we must be concerned with getting the amount of pulmonary ventilation necessary not only to get oxygen into the blood, but also to adequately clear the blood of carbon dioxide. I feel quite sure that we are going to see improvements in mechanical equipment and, by further evaluation of the actual job of ventilation being done, I think we are going to find that mechanical types of breathing will be of considerable value, especially in prolonged operations. I do not think we need worry about accumulation of carbon dioxide in short procedures; it is in the long procedure that we must be concerned.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—In my opinion it is a fundamental phase in intrathoracic surgery that has been touched upon by the previous speakers. Mechanical control of respiration as soon as the chest is opened, in our experience, is the only method that gives respiration similar to the physiologic. As you know, we have used this type of respiration on my service for more than fifteen years in all our clinical cases, with most satisfactory results. It was based upon laboratory studies undertaken in the early thirties. These showed that mechanically controlled respiration, with an inspiratory phase similar to the one the animal had when breathing spontaneously, and an expiratory phase without any increase of pressure so that the expiratory gas could flow out freely, gave the best results when investigating the arterial oxygen tension and the content of carbon dioxide in the blood, as well as the values of the pH.

We have used mechanically controlled respiration now in several thousand clinical cases, and we have had no complications from using this method. It has been very

gratifying to me to find that in this country during the last few years, an increasing interest has been shown in using this type of respiration for prolonged chest operations. I believe that the retarded development in this field in your country is due to the two schools of anesthesia from Madison and Boston, which emphasize that the anesthetist must follow the spontaneous respiration very closely in order to judge the condition of the patient. I am convinced, however, that there is no reason whatsoever not to paralyze all the respiratory muscles and to regulate the respiration of the patient mechanically during the time of an open chest operation, which method certainly is superior to any other.

DR. PAINE (closing).—I think one can appreciate the value of this valve in an experimental laboratory. We have had no dog die of anesthesia in the last 150, since this instrument has been used. We have been able to dispense with an attendant to take care of the anesthesia; the thing is set up, then one scrubs and goes ahead. We have been trying to get our anesthetists in Buffalo to use this clinically, with some reluctance on their part. Maybe they think they will soon not be needed. Probably 40 or 50 per cent oxygen would be better than 100 per cent.

I might just show you the fourth slide, which shows the interior of this little valve; it is an ingenious affair. Anyone who is interested can purchase this machine from the Mine Safety Appliance Company; they did have an office here in Denver.

THE DEVELOPMENT OF, AND CERTAIN CLINICAL APPLICATIONS FOR, A PORTABLE MASS SPECTROMETER

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MINNEAPOLIS, MINN.

RECENT technical advances have made possible an immediate assessment of the blood oxygen saturation and ether and oxygen concentrations in the inhaled gaseous mixture. These several devices supplement the anesthetist's sense of clinical judgment in the care of his patient. However, the instantaneous evaluation of alveolar carbon dioxide tension in the anesthetized patient still remains an area of confusion and recurring concern to the conscientious anesthetist, and more important, a grave potential hazard to the patient. Many attempts of limited success have been made to record the onset and duration of hypercapnia, but to date the interference introduced by those mixtures of anesthetic gases commonly used has prevented accurate, frequent, and rapid analyses for carbon dioxide. The problem of hypercapnia has recently been emphasized by both Beecher and Stormont.[1, 2] The former stressed the importance of this problem, particularly in thoracic surgery where the issue is often additionally complicated by an associated diseased respiratory system. His studies reported an average increase of the alveolar carbon dioxide concentration to 13.7 per cent in 16 pneumonectomy cases. This rise was associated with an average drop in the blood pH to 7.09. The Henderson-Hasselbalch equation was utilized for calculation of the alveolar carbon dioxide value after measuring the arterial blood pH and carbon dioxide concentration. Though such physicochemical methods are very accurate under normal conditions, the time involved in analyses and the necessity for anaerobic arterial blood samples as well as the errors introduced by the presence of anesthetic gases limit their widespread use as clinical tools for the anesthetist. The purpose of this paper is to present an alternative method for resolving certain of these difficulties.

At the University of Minnesota a continuously recording portable mass spectrometer has been developed by one of us (A. O. N.) for use in the operating rooms and physiologic laboratory as a rapid recording gas analyzer. This electronic device (Fig. 1) is capable of quantitatively analyzing the composition of a mixture containing five different gases. Every twenty seconds the process is repeated with an accuracy within 1 per cent. The presence of anesthetic gases introduces no measurable error. An instrument such as this should be an asset to the anesthetist, for it would allow him to obtain immediate knowledge of

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carbon dioxide concentration in the exhaled mixture as well as the amount of anesthetic gas in the system. He is therefore in a more favorable position to diagnose hypercapnia at its incipiency, to take the necessary corrective measures.

The mass spectrometer has been used for about thirty years by physicists in their study of the elements. It has been used extensively in industry as a leak detector and analyzer of unknown gas samples. However, biologists have been slow to adapt this method to their problems. It has served investigators as a stable isotope analyzer in the clarification of metabolic processes. The specific shortage of portable units and the relative unavailability of other models capable of analyzing a continuously changing gas sample has hampered work in this field. Except for the reports by Nier and associates[3] on continuous analysis of process stream contaminants in the Oak Ridge gaseous diffusion plant, and Kydd's study of CO_2/O_2 concentrations during alveolar carbon dioxide sampling,[4] no reports have appeared in the literature about a mass spectrometer capable of continuous gas analysis when the composition of the mixture is rapidly altered.

Fig. 1.—A picture of the mass spectrometer in the operating room.

The mass spectrometer used for the study being presented was designed to provide a portable instrument capable of rapid, continuous, automatic, quantitative recording of the components of the gas stream delivered to it. Fig. 2 illustrates schematically the basic features of this instrument. A sample of gas is drawn into an evacuated U-shaped chamber through a graduated leak and gaseous ions are formed by the electron beam. The trajectory of an ionized particle is dependent upon: (1) the molecular mass of the ionized particle, (2) the speed of the particle passing through the tube, (3) the amount of magnetic force acting upon the particle. With the magnetic force constant, the speed of specific particles passing through the tube is varied cyclically by

changing the difference of potential across the two slit plates. As a result of this cyclic variation, each component beam of ions arrives systematically at a common ion collector. The current carried by these ionic charges is then amplified so as to be recorded by a standard commercial six-point potentiometer such as is commonly used for recording temperatures.

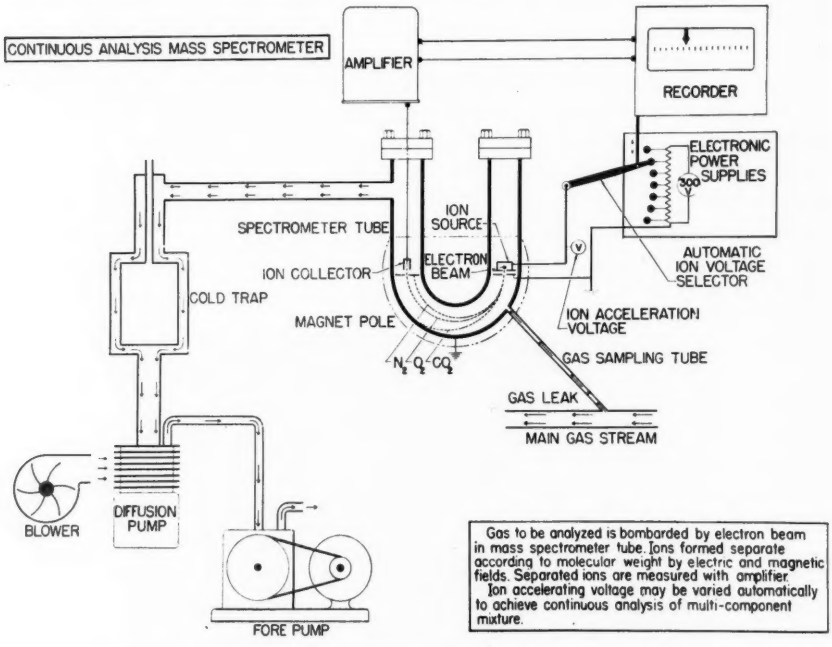
CONTINUOUS ANALYSIS MASS SPECTROMETER
AMPLIFIER
RECORDER
ELECTRONIC POWER SUPPLIES
300
SPECTROMETER TUBE
ION SOURCE
ELECTRON BEAM
ION COLLECTOR
AUTOMATIC ION VOLTAGE SELECTOR
COLD TRAP
MAGNET POLE
ION ACCELERATION VOLTAGE
N_2 O_2 CO_2
GAS SAMPLING TUBE
GAS LEAK
MAIN GAS STREAM
BLOWER
DIFFUSION PUMP
FORE PUMP
Gas to be analyzed is bombarded by electron beam in mass spectrometer tube. Ions formed separate according to molecular weight by electric and magnetic fields. Separated ions are measured with amplifier.
Ion accelerating voltage may be varied automatically to achieve continuous analysis of multi-component mixture.

Fig. 2.—A very much simplified schematic design of the basic principles of the action of the continuously recording mass spectrometer.

METHOD

The patients were anesthetized with ether, nitrous oxide, pentothal curare or a combination of all three agents. The preanesthetic medication was Nembutal, morphine sulfate, and atropine with the dosage regulated according to the age and physical condition of the patient. Thoracotomy cases were regularly placed in a semiprone position on a straight table. An intratracheal tube with an attached inflatable cuff was inserted so that manual ventilation could be performed. This assisted respiration was carried out intermittently in all of the cases anesthetized with pentothal curare, especially thoracotomy cases; here it was a routine procedure.

Following intubation the patient was connected to the mass spectrometer in the circuit shown schematically in Fig. 3. As illustrated, a continuous gaseous sample was withdrawn from the trachea—via a small bore polyethylene catheter threaded into and beyond the intratracheal tube to the vicinity of the carina. Circulation through this circuit was maintained by means of a small, leak proof,

air blower of sufficient capacity that the lag time between patient and spectrometer was about 90 seconds.

Prior to each testing period the mass spectrometer was calibrated against a gas of known composition; at hourly intervals during the procedure and at the conclusion of surgery, it was rechecked for possible drifting. Rarely was any significant variation detected.

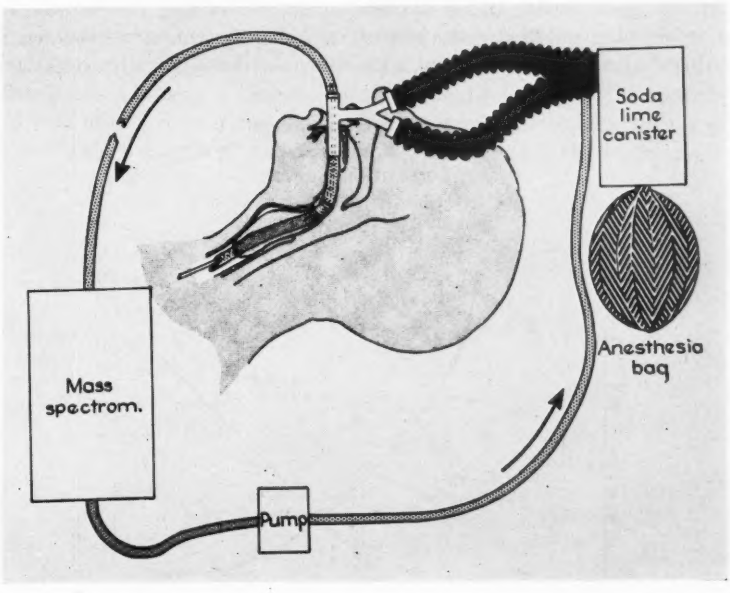

Fig. 3.—Schematic illustration of the sampling system used in the operating room to supply the mass spectrometer with a continuous stream of gas from the trachea of the patient.

An uninterrupted record rather than mere spot sampling was secured in each case. Peak accumulations of carbon dioxide and their related effects were more recognizable by virtue of an unbroken observation of these events. To avoid unsolicited cooperation from the anesthetist and to eliminate any explosion hazard the mass spectrometer was positioned at a distance of several feet from the anesthetic field. Since the toxicity of high alveolar carbon dioxide concentration probably rests chiefly on acidotic qualities, it seemed appropriate to correlate the carbon dioxide values with simultaneous blood pH levels. A control arterial sample was therefore drawn preoperatively under local anesthesia from the femoral artery. During the operative procedure a sample of blood was obtained either from an exposed artery in the operative field or from the radial artery. The pH determinations on these samples were made with a specially designed constant temperature glass electrode meter shown to be accurate to 0.01 pH units.[5] In the interim between sampling and analyzing, the blood was chilled to about 0° C. so that a reduction in pH due to glycolysis did not occur.[6]

RESULTS

In order to determine the accuracy of the mass spectrometer as a quantitative carbon dioxide analyzer and to evaluate the usefulness of the instrument as it was to be used, in indicating alveolar carbon dioxide concentration, preliminary experiments were performed in the laboratory. Two dogs were anesthetized with Nembutal and a continuous sample of respiratory gas discharged through the spectrometer in the manner illustrated in Fig. 3. In the absence of an interfering anesthetic gas the carbon dioxide concentrations could be checked periodically for agreement with a Scholander gas analysis of the same

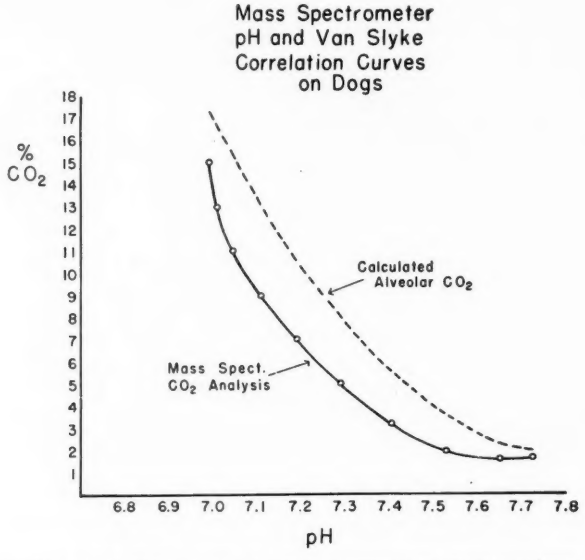

Fig. 4.—pH—CO_2 correlation curves of mass spectrometer recordings and simultaneous alveolar CO_2 per cent as calculated from arterial blood pH and CO_2 content

mixture.[7] Numerous tests were carried out and it was demonstrated that the two methods coincided within 0.5 per cent, that is, if the Scholander analysis gave a value of 10 per cent carbon dioxide the value obtained from the mass spectrometer would be between 9.5 and 10.5 per cent.

Comparison between the carbon dioxide values as recorded by the mass spectrometer using the sampling method outlined previously, and the alveolar air carbon dioxide concentrations were made possible by calculation of the latter value from arterial blood pH and total carbon dioxide content utilizing the Henderson-Hasselbalch equation.*

*In this equation with three interrelated variables, if any two of these become known the third can be readily calculated.[8] The equation is set up as follows, in order to determine carbon dioxide tension when the arterial pH and total carbon dioxide content have been measured (total carbon dioxide content measured by method of Van Slyke and Neill[9]):

$$pCO_2 = \frac{\text{Total } CO_2}{0.1316 \times 0.510\ (10^{pH - pK'} + 1)}$$

A respiratory acidosis due to excess carbon dioxide was induced in the dogs by removing the soda lime canister; alkalosis was produced by manual ventilation until a state of acapnia occurred. Arterial blood samples were drawn from the femoral artery during various stages of these procedures. Fig. 4 shows the variation of carbon dioxide concentrations and pH. The consistent difference between the mass spectrometer carbon dioxide concentration values and the calculated alveolar carbon dioxide concentrations, it seems reasonable to presume, is more likely an expression of the sampling technique used rather than any inherent error in the analysis. Because sampling is continuous throughout both inspiration and expiration, there is undoubtedly mixing of the true alveolar sample with dead space air and inspired air, whereas a truer alveolar air sample could be obtained only with elaborate sampling devices. Therefore, one would

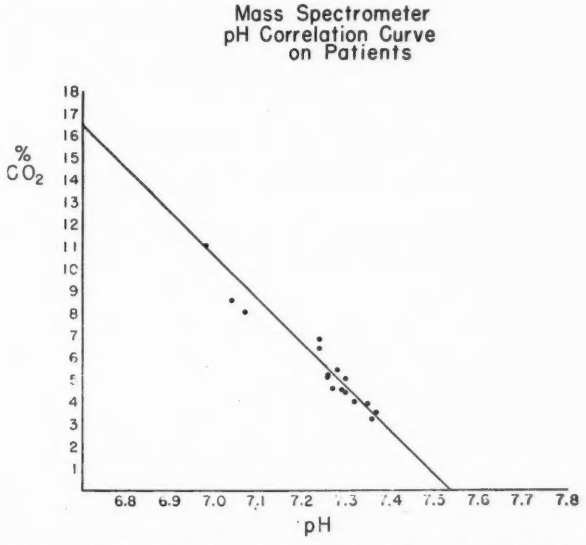

Fig. 5.—Tracheal air per cent CO_2 recorded by the mass spectrometer plotted against the pH of simultaneously drawn arterial blood from patients undergoing surgery.

predict that carbon dioxide concentration would be less in the mass spectrometer sample which is obtained partially from both phases of respiration. This is borne out by the results given in Fig. 4. The parallelism of contour between the two curves implies a correlation which should allow a close estimation of the alveolar carbon dioxide concentration when the mass spectrometer reading is known.

Fig. 5 illustrates the relation between the mass spectrometer records and some pH values obtained during surgery. The degree of accord is indicated by the interpolated line. Our inability to secure sufficient analyses with low arterial pH values hardly justifies any attempt at statistical treatment of these data. However, the real correlation seen from pH 7.37 to 7.25 permits the assumption that this method is a relatively accurate index.

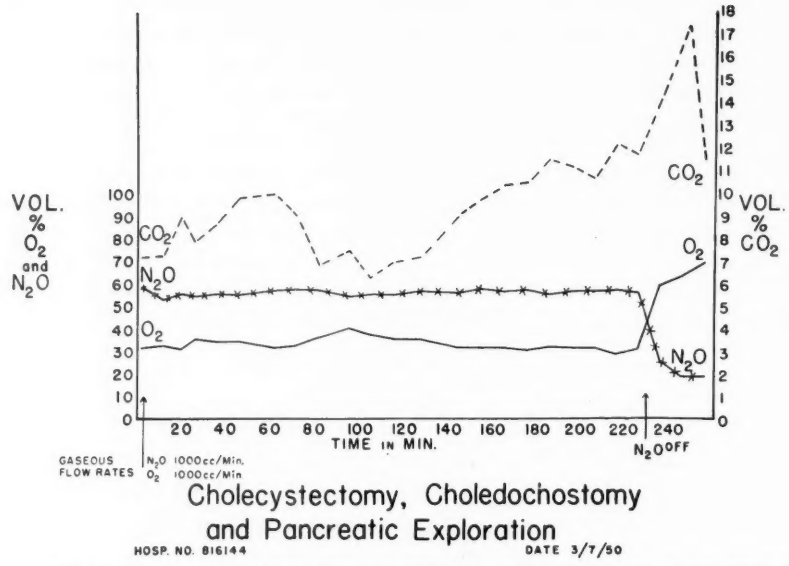

Fig. 6.—Results of a mass spectrometer recording. Note that the CO_2 values listed along the right ordinate are on a different scale from the O_2, N_2O values shown along the left ordinate. The small figures under the chart labelled N_2O and O_2 flow rates were values taken from the gas flow meters on the anesthesia machine.

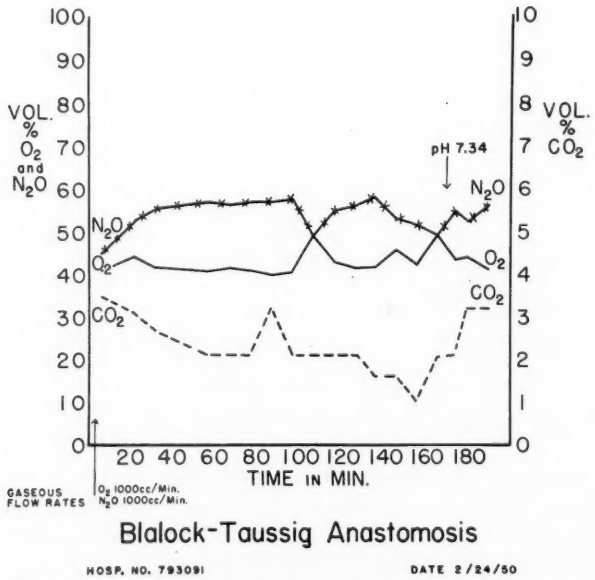

Fig. 7.—This replotted mass spectrometer record shows the low values encountered in a patient with compensated alkalosis. The arrow designates the point at which the arterial blood sample was drawn for pH analysis.

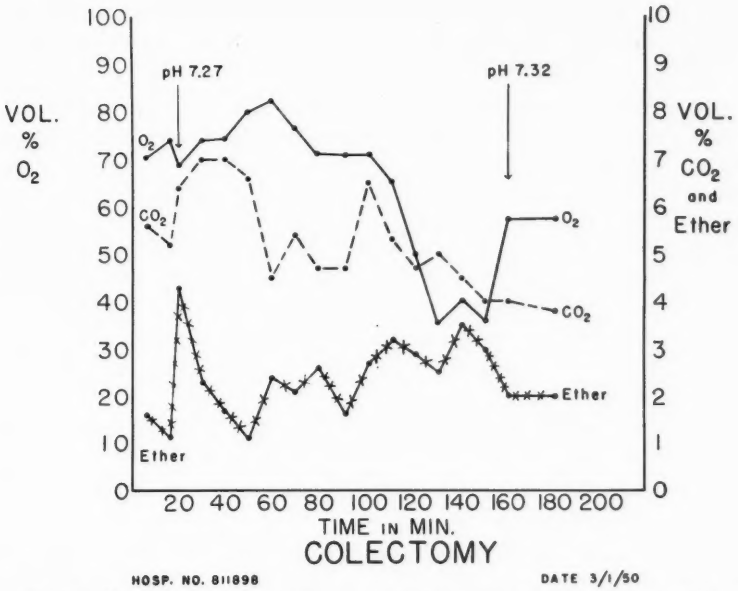

Fig. 8.—Mass spectrometer results on a patient anesthetized with ether. Note that the ether concentration is plotted on the same scale as the CO_2. For the most part the patient was carried with an ether concentration of 3 per cent or less. This is typical of most of the cases analyzed.

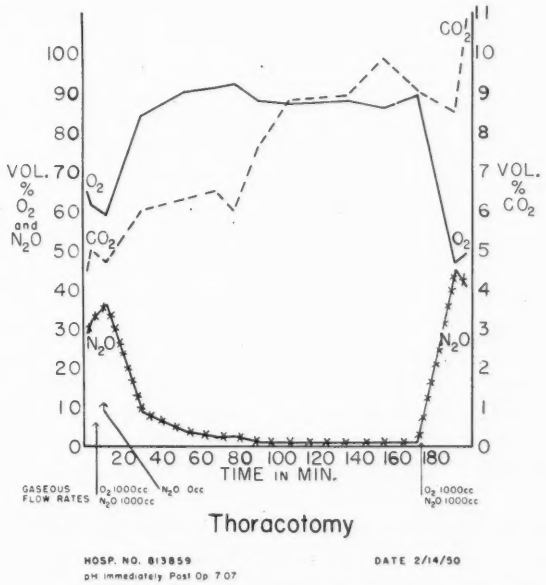

Fig. 9.—Mass spectrometer results on a thoracotomized patient. The neoplasm found was inoperable.

Figs. 6 through 10 are condensed reproductions of some of the recordings obtained. It should be remembered that 3 to 3.5 per cent carbon dioxide is a normal level for the sampling system employed.

On one occasion the carbon dioxide rose to 17.3 per cent, as is illustrated by Fig. 6. This is the highest value observed during the present series of experiments. Unfortunately no pH determinations were obtained, but by estimate from Figs. 4 and 5 it probably fell below 7.00. Following a common bile duct exploration and cholecystectomy, as this patient was moved to the recovery room, the blood pressure dropped to 80/60. This response was quite incompatible with the amount of blood lost and replaced during the operative procedure.

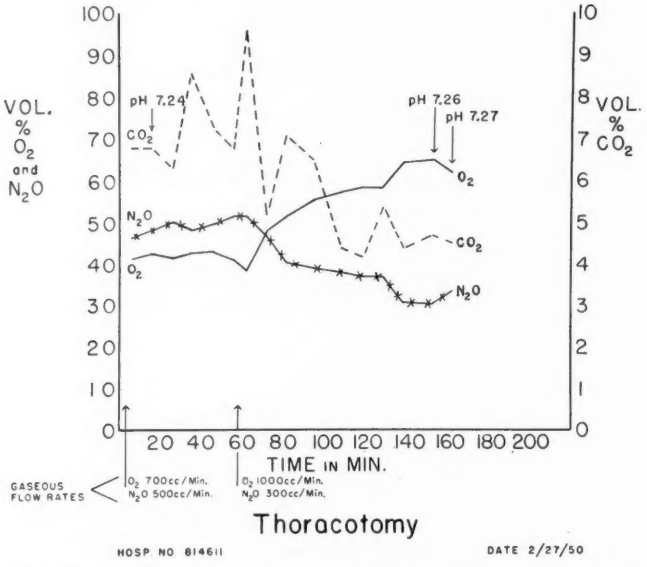

Fig. 10.—This illustrates another mass spectrometer tracing of a patient with a thoracotomy.

Hypotension persisted for 7 days, except when relieved periodically by ephedrine and neo-synephrine administration. During this period there was a diminished urine output, a rise in blood urea nitrogen to 77 mg. per cent on the fourth postoperative day, and a marked electrolytic imbalance. Serial eosinophil counts showed no diminution following an injection of epinephrine on the ninth postoperative day. On the seventh postoperative day the blood pressure rose to 110/60 and afterward stabilized above 100/70 as the patient gradually convalesced.

Table I presents a summary of the highest carbon dioxide concentrations recorded by the mass spectrometer for each case. The average was 7 per cent or approximately double the normal value. This suggests that at some time in these operative procedures the average case had an alveolar carbon dioxide con-

TABLE I. LARGEST CO_2 VALUES RECORDED BY MASS SPECTROMETER DURING THIRTY-FOUR OPERATIONS

PROCEDURE	ANESTHESIA	TOTAL NUMBER CASES	NUMBER OF CASES					AVERAGE LARGEST CO_2 CONCENTRATIONS
			LESS THAN 5% CO_2	5 TO 6% CO_2	6.5 TO 8% CO_2	8 TO 10% CO_2	10% CO_2 OR GREATER	
Pneumonectomies	Ether	1	–	–	–	–	1	10.0
Lobectomies	Pentothal, curare, N_2O	2	1	–	–	–	1	7.5
Miscellaneous Thoracotomies	Ether	1	–	1	–	–	–	5.2
	Pentothal, curare, N_2O	12	4	1	3	3	1	6.7
Laparotomies	Ether	10	5	2	3	–	–	5.6
	Pentothal, curare, N_2O	5	1	1	3	–	1	9.3
Miscellaneous	Ether	1	1	–	–	–	–	4.4
	Pentothal, curare, N_2O	2	–	1	–	1	–	7.0
Total	Ether	13	6	3	3	0	1	5.6
	Pentothal, curare, N_2O	21	6	3	6	4	3	7.7
	Composite	34	12	6	9	4	4	7.0

centration of about 10 per cent and that this situation was associated with an arterial pH of approximately 7.20.

DISCUSSION

The correlation data shown in Figs. 4 and 5 reveal a definite pattern between the alveolar carbon dioxide samples as estimated by the Henderson-Hasselbalch equation and those values recorded by the mass spectrometer. The latter are uniformly lower. The high reproducibility between the mass spectrometer and chemical gas analyses, earlier referred to, confirm the likelihood that the gas sampled by the mass spectrometer did not represent true alveolar gas. It is difficult to deliver a true alveolar sample to the analyzer without interfering with anesthetic techniques. It was necessary therefore to standardize the instrument and the involved setup by biologic testing and extrapolation from this for clinical material. The results shown in Fig. 5 indicate that the normal carbon dioxide value was 3 to 3.5 per cent.

In so far as the acid base balance was concerned, this method is only applicable in assaying an uncompensated respiratory acidosis or alkalosis in which the alteration of pH is due solely to either an increase or decrease in carbon dioxide tension as the result of change in alveolar carbon dioxide concentrations. In a metabolic type of acidosis with the removal of fixed base or with the addition of fixed acid, the pH diminution is not due to an increased alveolar carbon dioxide tension, and the acidosis assuredly would not be revealable by the mass spectrometer. Beecher's concept[1] of a metabolic acidosis with an accumulation of fixed acid secondary to prolonged hypercapnia therefore introduces another unknown capable of altering the pH-CO_2 relationship. This situation with several vectors is only partially solvable by data from the mass spectrometer.

A limitation of the mass spectrometer-pH correlation data appears in the states of compensated respiratory acidosis or alkalosis. Here the fixed base of the body has been adjusted in order to compensate for the respiratory maladjustment. For example, in an emphysematous individual if one were to judge from only an elevated alveolar carbon dioxide concentration, it would seem that the patient was in a state of acidosis due to carbon dioxide accumulation. However, pH determinations might well show normal values because of the retention of base by the kidney so as to effectively neutralize the acidotic tendency. An example of this type of error is seen in Fig. 7, the chart of a case of tetralogy of Fallot on whom a Blalock-Taussig anastomosis was performed. It will be noted that the carbon dioxide values were consistently under 3 per cent. Under ordinary circumstances one would assume this to be due to overventilation of the patient by the anesthetist, leading to a respiratory alkalosis. However, pH determinations performed when the mass spectrometer reading was 2 per cent showed a slight acidosis. An attempt to raise the alveolar carbon dioxide concentration by hypoventilation would have aggravated the acidosis. The explanation hinges about the customary respiratory alkalosis these individuals carry secondary to chronic hyperventilation as described and analyzed by Bing and associates.[10] Here the kidney compensates for the diminished alveolar carbon dioxide tension by excreting base so as to hold the pH at 7.40. Therefore, in the case illustrated, when the mass spectrometer recorded a level of 3 per cent, which is normal for most cases, the child would undoubtedly have shown a pH depression, had an analysis been performed at that time, because the amount of base available would be insufficient to neutralize the accumulated carbon dioxide. Fortunately this discrepancy between pH-CO_2 values due to preoperative compensation can be corrected by drawing an arterial sample sometime preceding surgery and analyzing it for total carbon dioxide content and pH. Consistently low carbon dioxide records in association with diminished pH values have been obtained in three cases of this type. However, in all of these cases the carbon dioxide analyses were also complicated by the fact that comparable intratracheal samples could not be obtained due to impedance of the airway by the sampling tube. Accordingly these cases have not been included in the pH-CO_2 correlation curve (Fig. 5).

Mass spectrometer determinations made during ordinary operative procedures are valid since the compensating mechanism of base excretion or retention by the kidney lags. When one sees, therefore, a rise or fall of carbon dioxide concentration during an operation it is virtually inconceivable that kidney compensating mechanisms account for this but rather it represents an actual shift of acid-base balance.

A rise in blood pressure has been the common sign accepted by anesthetists as signaling the presence of carbon dioxide excess. During any major surgical procedure so many variables are present to interfere with the blood pressure as to make it extremely unreliable as a diagnostic sign of hypercapnia. This has been substantiated by the carbon dioxide analyses. In only a few instances was there a rise in blood pressure coincident with carbon dioxide elevation.

While carbon dioxide excess in the ranges observed is normally a marked respiratory stimulant, the anesthetic agents so effectively interfered with the function of the respiratory center that an increase in respiratory rate or volume was seldom noticed in conjunction with high carbon dioxide levels.

The toxic effects of carbon dioxide in asphyxia are well documented.[11] The depressant effect of high inhaled carbon dioxide concentrations on the respiratory center are also well known.[12] The narcotizing effect of carbon

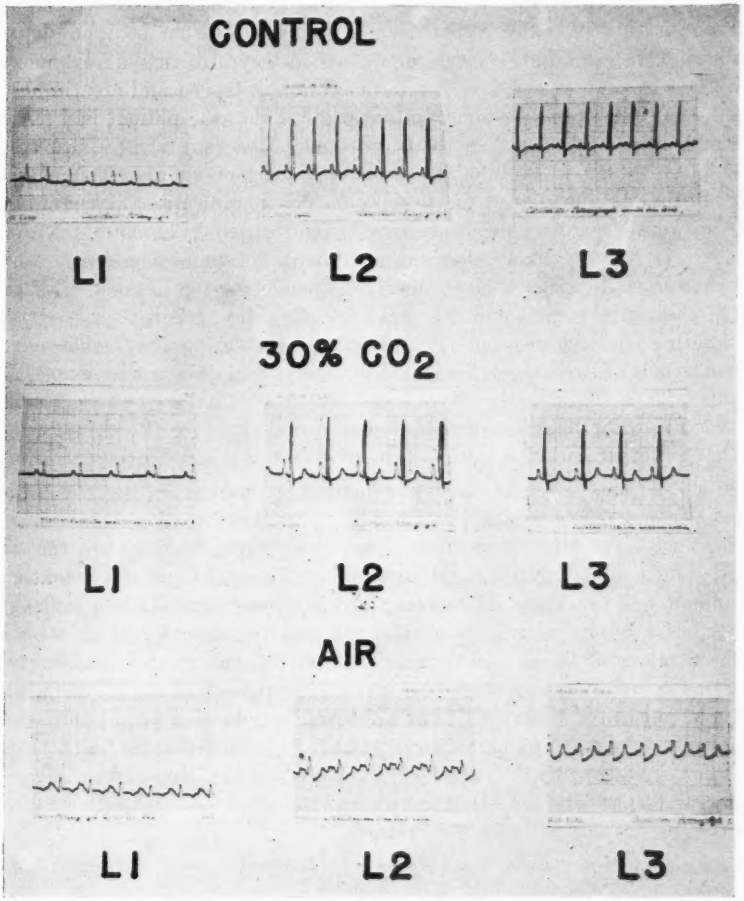

Fig. 11.—Electrocardiographic tracings of anesthetized dogs taken before, during, and after breathing 30 per cent CO_2/O_2 mixtures.

dioxide inhalation has been demonstrated with its use as an anesthetic agent.[13] Waters[14] described one postanesthetic death which he stated was most likely the result of hypercapnia. Indeed it seems a reasonable hypothesis that some of the unexplained grave operative sequelae, including cardiac standstill, are connected with this problem.

The postoperative complications in the instance in which the carbon dioxide level reached 17.3 per cent introduce interesting speculative possibilities in association with certain laboratory investigations. It has been demonstrated in the dog that alveolar carbon dioxide concentrations above 15 per cent produce depression of adrenal function as evidenced by the absence of an eosinopenia following the injection of epinephrine.[15] The duration of this effect after cessation of the hypercapnia awaits further studies. Depressed adrenal function conceivably could account for most of the following postoperative complications observed in this patient: hypotension, diminished serum sodium, depressed carbon dioxide combining power, depressed kidney function as evidenced by diminished urinary output and rise in blood urea nitrogen and creatinine. Another factor which must be taken into account is that the patient had markedly abnormal liver function tests both pre- and postoperatively. Seevers and Orth have shown that inhalation of high carbon dioxide concentrations probably produces injury to the liver.[16, 17] The complications observed in this case, including the low blood pressure, would also be compatible with liver damage. It is quite likely that carbon dioxide accumulation during surgery may have been the trigger mechanism responsible for the depression of one or both of these vital organs and may have provoked the serious sequelae outlined.

Studies are underway in this laboratory evaluating the toxicity of high concentrations of carbon dioxide in the inspired air of anesthetized experimental animals. So far these studies have indicated changes along the following lines: (1) electrocardiographic evidences of myocardial damage, (2) decreased tolerance to pentothal and Nembutal, (3) marked arterial blood pH depressions.

Fig. 11 shows a set of electrocardiographic tracings during the course of an experiment. This record is typical of a number of others that have been obtained with the same procedure. The upper three tracings are the control records taken following pentothal anesthesia. The middle set were taken while the animal was breathing 30 per cent carbon dioxide and 70 per cent oxygen, and the last group represents a time interval ten minutes after the animal had been taken off the carbon dioxide mixture and returned to breathing room air. In the latter tracing there is a marked depression of the ST segment suggestive of myocardial anoxia. That a marked hypotension occurs following the cessation of inhalation of high concentrations of carbon dioxide has been shown by other investigators.[18] Whether the hypotension causes the myocardial changes evidenced by the electrocardiographic records or whether the reverse situation occurs has not been ascertained.

Another phase of these studies was the demonstration of decreased tolerance to pentothal and Nembutal. A marked blood pressure fall and bradycardia were noted after administration of the drugs following a period of inhaling mixtures of high carbon dioxide content. After a period of breathing 30 per cent carbon dioxide and 70 per cent oxygen three out of ten dogs died suddenly subsequent to the administration of pentothal or Nembutal doses ordinarily readily tolerated by normal dogs. Beecher and Moyer have also demonstrated a decreased tolerance to barbiturates in the presence of respiratory acidosis.[19]

Inhalation of 30 per cent carbon dioxide caused a pH depression as low as 6.61 in some of the dogs.[20] This is considerably under the level of 6.80 to 7.00 commonly acknowledged in standard textbooks of physiology to be the lower limit compatible with life. Surprising as it is that these dogs survived, it is perhaps no more important than the warning that such high alveolar carbon dioxide concentrations produce such critical arterial pH values. It is to be emphasized that these were acute experiments and no attempt was made to examine the postacidotic sequelae. Furthermore the tolerance of healthy mongrel dogs to acidosis is likely to be disproportionately more elastic than for patients assaulted by chronic disease and the stresses of both anesthesia and major surgery.

SUMMARY

A portable mass spectrometer functioning as a continuously recording gas analyzer is described. The clinical adaptation of the instrument has been noted, with reference to its future in the operating room as an aid to the anesthetist in ascertaining the carbon dioxide concentration in expired air. Graphs are shown of the results with five typical cases. These graphs also show concentrations of oxygen and anesthetic gas in the sample.

The correlation of the carbon dioxide concentration and arterial blood pH are shown both in experimental animals and patients. These results indicate a reasonable agreement between two methods of analysis.

This instrument has tested quantitatively the concentration of exhaled gases during thirty-four major operative procedures, including many transthoracic procedures. It is demonstrated that on the average a level of 6.8 per cent carbon dioxide was reached at one time or another during most of those cases. This corresponds to an arterial blood pH of 7.20 and an alveolar carbon dioxide concentration of approximately 10 per cent.

The postoperative complications in one case in which the carbon dioxide level reached 17.3 per cent are reviewed.

Results are reported from carbon dioxide toxicity experiments carried out on experimental animals.

The authors gratefully acknowledge the technical assistance of Mr. Howard Demorest and Miss Margaret Murray.

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THE SURGICAL TREATMENT OF GIANT EMPHYSEMATOUS BLEBS AND PULMONARY TENSION CYSTS

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IN 1925 Koontz[1] reported the first case of emphysematous blebs in the American literature. Shortly thereafter, Miller[2] attempted to clarify the differential pathology between blebs and bullae, and was further of the opinion that these localized abnormalities were always associated with a generalized emphysema. Miller described a bleb as a subpleural collection of air caused by rupture of pulmonary alveoli immediately beneath the pleura allowing the air to escape into the pleura forming a thin-walled bladder-like prominence outside the lung itself. He defined a bulla as a vesicular emphysema situated within the lung, and though it often projected beyond the surface of the lung, it was covered by an intact pleura. In the subsequent literature many synonyms have been used: pneumatocele, cystic emphysema, air cyst, bullous emphysema, emphysematous blebs, and vanishing lung; all of these terms conform to Miller's original concept of subpleural blebs.

Study of our own surgical material indicates that we removed blebs only, but for practical purposes there does not seem to be great merit in attempting to distinguish between blebs and bullae. In some cases, we have removed localized blebs from lungs which grossly, at least, showed no evidence of emphysema. Extended inquiry into the pathogenesis of pulmonary emphysema is not germane to this presentation. Differentiation, however, should be made between emphysematous blebs and intrapulmonary cysts. Much of the literature is confusing. In our opinion, pulmonary cysts are congenital in origin, have a definite fibrous tissue wall, and are lined by epithelium. Emphysematous blebs are acquired, perhaps very early in life and have either no lining or at most have an alveolar type of flattened epithelium.

Why these lesions develop is not at once apparent nor can it be determined why all patients with emphysema do not develop large air cysts. Suffice it to say that the end result in many patients is a great decrease in pulmonary function with symptoms ranging from mild dyspnea to severe incapacitation. Definite therapy therefore becomes necessary.

METHODS OF THERAPY

No one familiar with this disease questions the necessity for treatment of individuals presenting severely incapacitating or life-threatening symptoms from bullous emphysema. The therapeutic approach has been the subject of

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considerable controversy and authoritative sources recommend many measures varying from a "conservative" regime of observation to extensive excisional surgery. Spontaneous disappearance of an emphysematous cyst without active treatment of any kind has been reported.[3, 4, 5] This phenomenon has been explained on the basis of rupture, absorption of air, and gradual expansion of the previously compressed lung. These remarkable cures have been reported in infants under two years of age for the most part but, unfortunately, are the exception. Needle aspirations of the air-containing sac has likewise resulted in complete clearing.[4, 6, 7, 8] The danger, however, of sudden exodus by needle rupture of a cyst has long been recognized[9, 10, 11] and such a procedure should never be attempted without facilities for immediate thoracotomy. In line with this same therapeutic approach, the injection of Lipiodol and caustics has been advanced. King and Harris[12] reported the successful treatment of a congenital lung cyst by intracavitary injections of iodized oil with subsequent closure of the bronchial communications but at the same time (1937) predicted that excisional surgery would ultimately be the most effective treatment. Hennell[13] had a similar experience with Lipiodol. Silver nitrate cauterization of a cyst wall following open drainage was reported by Pierson,[14] in 1935, and again two years later by Field and Rosenberg.[15] In 1928, Eloesser[16] felt quite strongly that only infected cysts should be treated and these he drained by his flap technique following later with a plastic procedure to close the opening in the chest wall. In 1931, Eloesser[17] added subsequent lobectomy with closure of the fistula to his primary drainage operation. Anderson,[18] in 1937, likewise felt that drainage of infected cysts was the preferred treatment. Brown and Brock,[19] in 1942, advocated the endocutaneous flap procedure for the treatment of a large cyst and in their report described the technique of exteriorization of the cyst wall. These early therapeutic endeavors were during the period when thoractomy was hazardous and represented a compromise with surgical principles of intrathoracic exploration as we know them today. Such historical attempts are for the most part discarded procedures at the present time.

Nissen,[20] in 1945, advocated thoracotomy, direct opening of the cyst, and repair by a plication-inversion suture technique. This procedure was performed in two cases with ultimate success, although there was delay of several weeks in the re-expansion of the affected lung. Head and Avery[21] presented the technique of decompression of an emphysematous bleb by intracavitary suction identical with Monaldi drainage and provided an excellent approach to the problem. In their series of eight cases good results followed this minor procedure under local anesthesia. They emphasized the applicability of suction therapy to the patient whose vital reserve is dangerously reduced because of high intracavitary pressure. We can best stress, however, that at thoracotomy these pressures are removed as soon as the chest is opened and the patient's condition routinely and immediately improves to essentially a normal status as far as oxygenation is concerned. Head and Avery believe the uniform absence of pleural adhesions in their cases was possibly a coopera-

tive factor in the development of the blebs. This drainage method also was advocated by Lloyd[7] and may be well suited to patients with but a single giant bleb. It should be of little value, however, in the presence of multiple blebs which was the situation in many of our cases. Unfortunately, the roentgen examination frequently cannot tell us whether the blebs are single or multiple.

During the past ten years there has been an ever increasing number of essayists strongly favoring excisional surgery in the approach to this problem. Brock,[22] Ravitch and Hardy,[23] and Naclerio and Langer[24] reported the largest number of cases treated by resection but many other surgeons have been sponsors of this approach.[10, 25-28, 29-31] Burnett[32] and associates stressed the importance of careful selection of surgical cases and were most optimistic in those with well localized disease. Clagett[33] favored resection with utilization of segmental removal in suitable cases.

DIAGNOSIS AND SELECTION OF CASES

In the past three years, we have operated upon fourteen patients with giant emphysematous blebs by lobectomy, segmental resection, or local excision of the tissue involved. The cases subjected to surgery have been carefully selected. The importance of painstaking evaluation of the individual surgical candidate cannot be overemphasized. As is true in all types of surgical procedures, the best results are obtained in the younger age group although such fact does not preclude surgery in the older individuals if legitimate indications exist. The extremes in our cases were three months and sixty years of age. All of the patients presented definite symptomatology referrable to the chest, the presenting complaint unanimously being increasing dyspnea. All, with the exception of the infants, were faced with an immediate serious occupational handicap.

The two most important considerations are the symptomatology and the roentgen appearance. The prospective surgical patient usually presents the history of increasing dyspnea, pain in the chest, and frequent respiratory infections. He may also complain of slight dysphagia, cough, a feeling of pressure in the chest, and general fatigue. Physical examination in general reveals diminution of excursion over the involved area with decreased breath sounds. The roentgen appearance is essential in the diagnosis and is the most important single finding in the localization of the lesions to one or both lungs. Bilateral disease is not a contraindication to surgery but a unilateral lesion is much more encouraging. The pathologic area appears black and lacking in lung elements. In many instances the roentgen film gives the impression of a localized pneumothorax, the increased transparency of the lung being due to actual loss of vascular markings. The surrounding lung appears compressed and in those areas the vascular markings are increased. The lesions are for the most part located in the basilar areas although apical lesions of this type are common. If serial roentgenograms are available there is usually evidence of progression in size. Bronchoscopic examination reveals twisting and distortion of the bronchial orifices so that the normal relationship is disturbed. Lipiodol bronchograms are routinely produced to rule out the possibility of

associated bronchiectasis. Bronchography is of further aid in determining the amount of normal lung tissue that can be depended upon for re-expansion as well as to forecast in advance of surgery the type of resection which will be required. Bronchospirometric readings and vital capacity studies were not performed in our surgical cases.

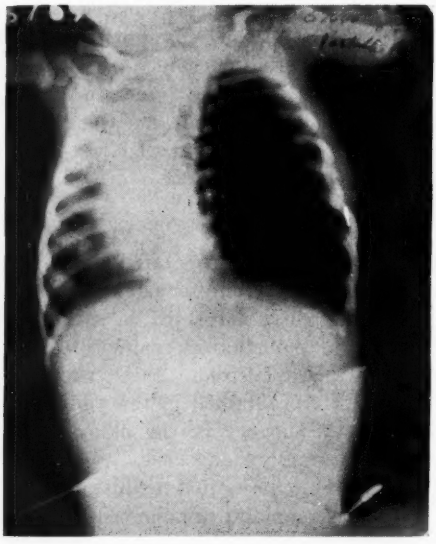


Fig. 1 (Case 1).—Roentgenogram taken immediately before left thoracotomy.

CASE REPORTS

CASE 1 (P. B.).—This 3-month-old infant girl was brought to her family physician on Aug. 5, 1948. For the preceding two months the parents had noted increased respirations and right-sided heartbeat. Roentgen examination revealed a displacement of the mediastinum and heart far to the right and a large emphysematous area occupying the entire left chest (Fig. 1). The patient was hospitalized and needle aspiration and intracavitary drainage were unsuccessful. Left thoracotomy was performed on Aug. 26, 1948, and immediately, when the pleura was opened, a giant emphysematous bleb eviscerated through the wound and was seen to occupy the entire lower lobe exclusive of the superior segment (Fig. 2). As soon as the chest was opened, the patient's condition improved. The left upper lobe was atelectatic, displaced in the upper posterior mediastinum and to the right of the vertebrae. An anatomic resection of the basal segment of the left lower lobe was performed. The postoperative course was satisfactory except for the development of an osteomyelitis of a rib end at the site of the drainage tube. This healed per primam after local resection. A ten-month follow-up showed a normal appearing chest (Fig. 3). Today the child is clinically normal.

CASE 2 (H. W.).—This second case demonstrates the value of early removal of emphysematous blebs in anticipation of the possibility of secondary infection and abscess formation. This 48-year-old man was first seen by us on July 9, 1948, with a history of having an "air pocket" in his right lung since 1945. Periodic respiratory infections had occurred for the past three years and on this hospital admission he complained of general malaise,

cough, and low-grade fever. Roentgen examination of the chest revealed a large emphysematous bleb occupying the right apex with an associated pneumonitis (Fig. 4). Comparison with radiographs made in 1945 showed definite evidence of progression. Bronchoscopic drainage relieved the cough and pyrexia and the patient elected to postpone surgery. One week later a fluid level developed within the emphysematous area (Fig. 5) and the patient submitted to operation. At thoracotomy a large emphysematous bleb which occupied the entire right apex was removed. At its upper and lower lobe attachments it was

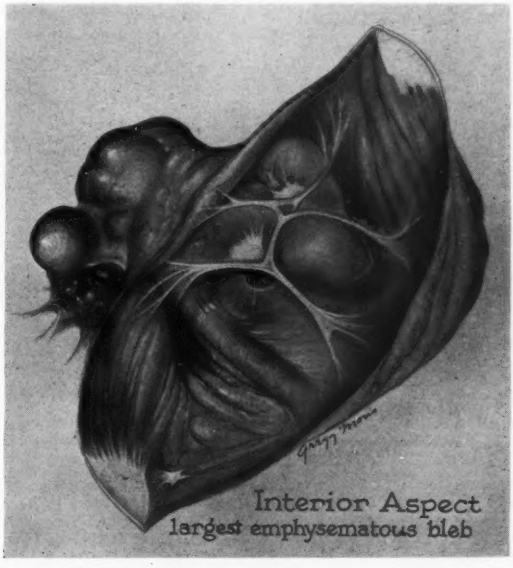


Fig. 2 (Case 1).—Schematic drawing of left lower lobe bleb occupying basal segments.

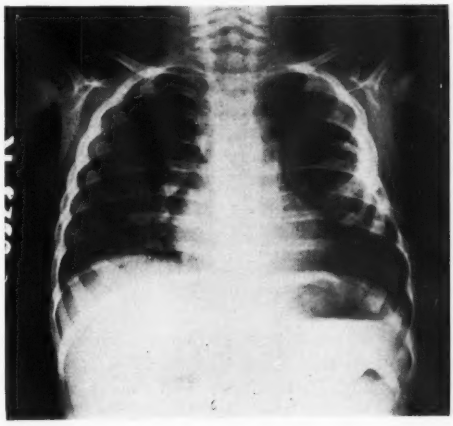


Fig. 3 (Case 1).—Roentgenogram taken ten months following resection of basal segments of left lower lobe for large emphysematous cyst.

necessary to perform wedge resections to avoid rupture of the cystic mass. The surgical specimen revealed a definite abscess cavity in the lower lobe attachment (Fig. 6). The patient had an uneventful postoperative course and when last seen eighteen months following surgery, his cough had disappeared and he was free of symptoms.

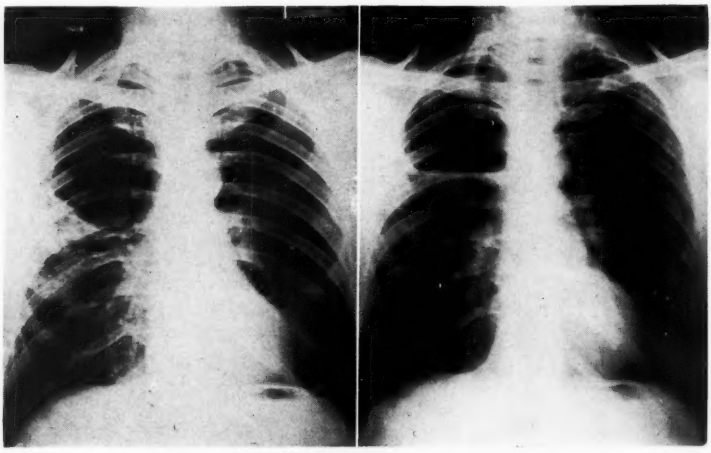


Fig. 4 (Case 2).—Radiograph taken three years after original examination demonstrating progression in size of bleb in right apex and development of associated pneumonitis.

Fig. 5 (Case 2).—Roentgenogram one week after Fig. 4 showing fluid level within emphysematous bleb.

Fig. 6 (Case 2).—Photograph of emphysematous bleb with concomitant pulmonary abscess right upper lobe.

CASE 3 (F. J.).—This 37-year-old man's symptoms were of one year's duration and he sought medical advice only because of mild exertional dyspnea and because he was told that he had a "spot on his lung." Roentgen chest examination in July, 1948, showed an area free of lung markings in the lower left chest (Fig. 7) and the remainder of the physical examination was without positive findings. Bronchoscopy and bronchography were noncontributory except that the Lipiodol failed to fill entirely the lingula of the

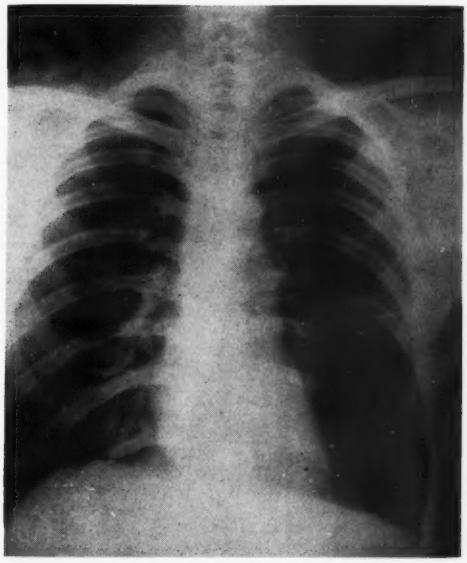


Fig. 7 (Case 3).—Roentgenogram showing localized emphysematous area left base.

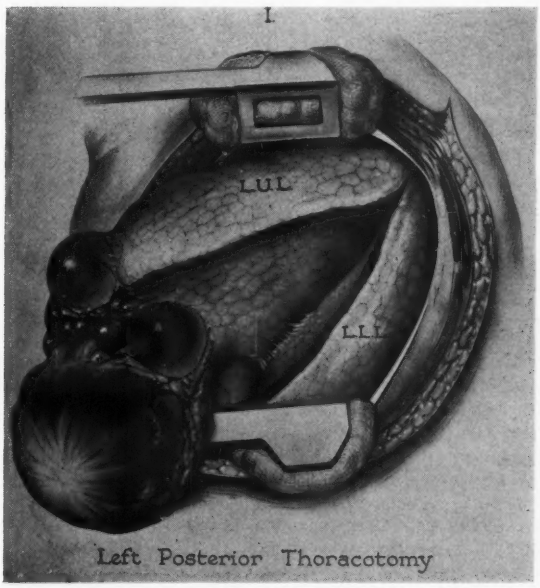


Fig. 8 (Case 3).—Artist's interpretation of left lung at operation with cystic emphysema confined to lingula. The remainder of the left upper lobe was grossly normal.

left upper lobe. Thoracotomy was performed on Dec. 31, 1948, and a large emphysematous bleb was found to occupy the entire lingular segment of the left upper lobe (Fig. 8). An anatomic segmental resection of the lingula was performed and the patient was discharged without event. A follow-up study one year later revealed no abnormality and the patient was without chest complaints.

CASE 4 (R. W.).—This male infant, 8 months of age, was admitted to the hospital because of progressive weight loss, inability to take his feedings, and asthmatic symptoms of three months' duration. Physical examination revealed obvious respiratory distress. There was absence of breath sounds in the left chest and the heart was shifted to the right. Chest roentgenogram confirmed the physical findings and showed what appeared to be a giant cyst occupying the entire left hemithorax (Fig. 9). At thoracotomy on June 7, 1949, a giant bleb eviscerated through the wound and was found to be attached to the apical segment of the left upper lobe (Fig. 10). Local excision was performed and the upper and lower lobes expanded satisfactorily. Small intralobular emphysematous spaces were observed in the expanded upper lobe but this was not of sufficient moment to warrant additional upper lobectomy. The child's course was uneventful and when last seen on March 28, 1950, was entirely asymptomatic and chest roentgenogram was satisfactory.

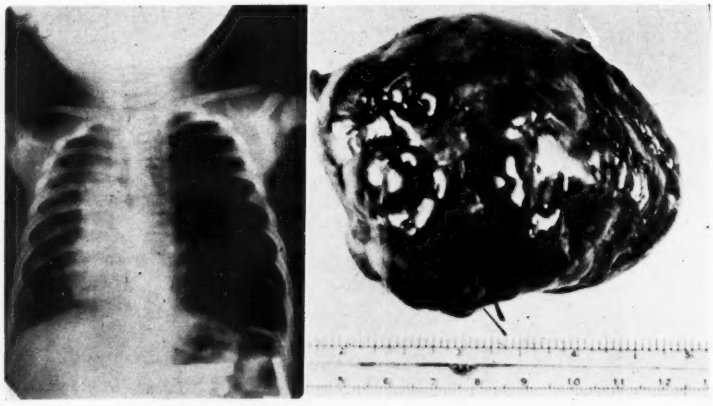
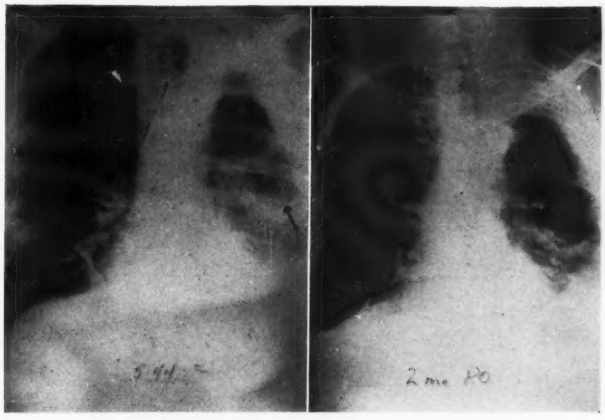


Fig. 9 (Case 4).—Roentgenogram showing complete absence of pulmonary markings on the left hemithorax due to compression by subpleural emphysematous cyst originating in apex of left upper lobe.

Fig. 10 (Case 4).—Photograph of surgical specimen.

CASE 5 (A. M.).—This 51-year-old man was first seen by us in November, 1946. His thoracic difficulties began in May, 1942, when he had a left spontaneous pneumothorax. He developed a hemorrhagic pleural effusion and was treated with needle aspiration over a period of several months. He was not seen again medically until 1946. His complaints were those of increasing dyspnea and left thoracic pain. Radiographic examinations of his chest showed a compressed emphysematous left lung covered with a thick pleural "peel" with definite cystlike areas in the upper and lateral portions of the right lung (Fig. 11*A*). This patient was obviously a pulmonary invalid and it was agreed that surgery of some type should be attempted. Feeling that a primary decortication would be required on the left before the blebs could be reached and further that the salvage value of the left lung was questionable, a right thoracotomy was performed on Nov. 5, 1946. Fig. 11B demonstrates the postoperative roentgenogram showing return of mediastinum and trachea to midline. Five separate subpleural blebs attached to the right upper and middle lobes were



A. *B.*

Fig. 11 (Case 5).—*A*, Preoperative roentgenogram showing overdistention of right lung, tracheal shift to left, and emphysematous area with fibrous compression of left lung.

B, Film taken two months following right thoracotomy and excision of five separate emphysematous blebs. The trachea is now in the midline proving that it was being *pushed* to the left because of increased pressure on the right, rather than being *pulled* to the left because of retraction on the left.

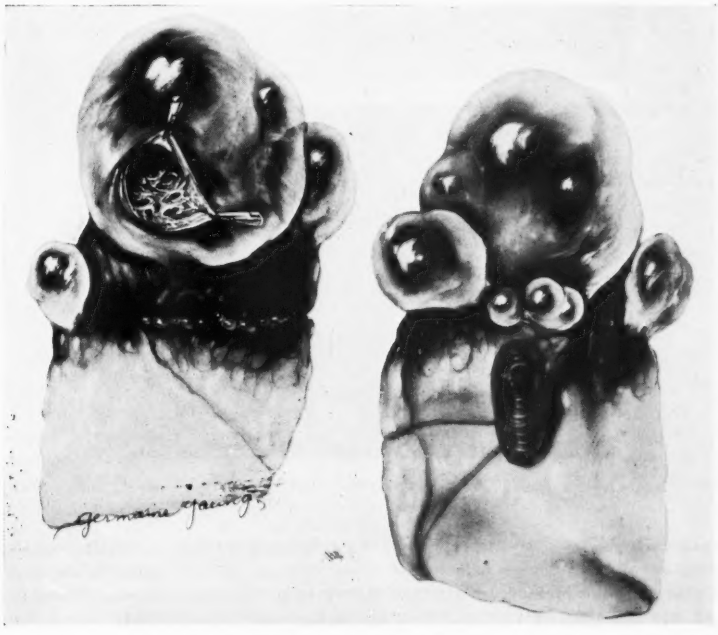


Fig. 12 (Case 5).—Schematic representation of gross appearance and location of subpleural blebs of right lung seen at thoracotomy.

locally excised without sacrifice of any functioning pulmonary tissue (Fig. 12). This patient's convalescence was complicated only by his hesitancy in returning to activity because of fear. However, when last seen approximately three years following surgery he was gainfully employed as a bartender. His dyspnea has not entirely disappeared and he still complains of pains in the left chest but he is able to make a living and is no longer an invalid.

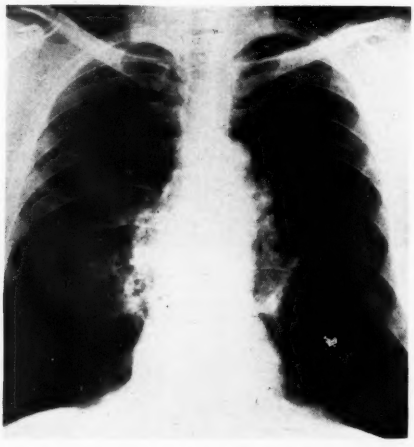


Fig. 13 (Case 6).—Radiologic examination of chest showing marked progression of bilateral cystic emphysema in period of two years.

Fig. 14 (Case 6).—Photograph of left lower lobe and lingula removed at time of surgery.

CASE 6 (B. M.).—This case represents the attempt to salvage an elderly individual who was totally invalided from his disease and is our only operative fatality in the series. He was 58 years old and presented a six-year history of progressive dyspnea. When first seen by us on April 3, 1949, he was extremely dyspneic, unable to walk more than a few steps, and would become cyanotic at the least exertion. He had been bedridden for four years and it was necessary for him to have oxygen available at all times. Serial roentgenograms showed large emphysematous areas at both bases with definite signs of progression

over the six-year period. The current radiograph is shown in Fig. 13. Fluoroscopic examination gave the impression that the greater involvement was on the left side and realizing the serious risk, a left-sided thoracotomy was performed on April 29, 1949. As large blebs replaced the entire left lower lobe and lingula (Fig. 14) a left lower lobectomy and lingulectomy were performed. The patient did well during the surgical procedure but developed progressive postoperative dyspnea and died twelve hours after return to his room. Autopsy showed well-expanded left upper lobe and enormous distention of the right-sided blebs, apparently due to mediastinal shift and increased pressure (Fig. 15).

In retrospect the outcome might have been favorable had operative provision been made for catheter drainage on the right prior to left-sided thoracotomy.

Fig. 15 (Case 6).—Photograph of autopsy specimen of overdistended emphysematous belbs in contralateral lung.

CASE 7 (L. G.).—This 42-year-old man was first seen regarding his chest complaints on June 21, 1949, and while he had noticed mild dyspnea for several years previously, this complaint had become so much greater recently that he was urgently seeking help. Roentgen chest examination showed bilateral emphysematous areas at both bases (Fig. 16). General physical condition was good and right thoracotomy on July 29, 1949, presented multiple thin-walled cysts on all three lobes. These were all locally excised. The immediate postoperative course was stormy due to bronchial plugging and increased distension of the bullous disease on the left. The patient responded to bronchoscopy and needle aspiration of one of the left-sided cysts. He was readmitted on March 5, 1950, for treatment of the left lung and reported no dyspnea but minimal cough. Chest radiograph showed good expansion of the operated side (Fig. 17) and on March 7, 1950, a left thoracotomy was performed with excision of numerous peripheral blebs of the upper and lower lobes. On this occasion his convalescence was entirely without difficulty and films taken on March 24, 1950, show excellent expansion of the left lung (Fig. 18).

CASE 8 (H. M.).—This 51-year-old man was seen in December, 1946, during a pneumonic process involving the right upper lobe. He cleared promptly with penicillin and further investigation revealed a 10-year history of dyspnea with accentuation in the past eighteen months. He had also experienced bouts of pneumonia yearly for the previous five years and a thoracic roentgenogram showed multiple areas of what appeared to be subpleural blebs over the right upper lobe and along the mediastinum. On Jan. 28, 1947, a right thoracotomy was performed and seven separate blebs were locally excised from the

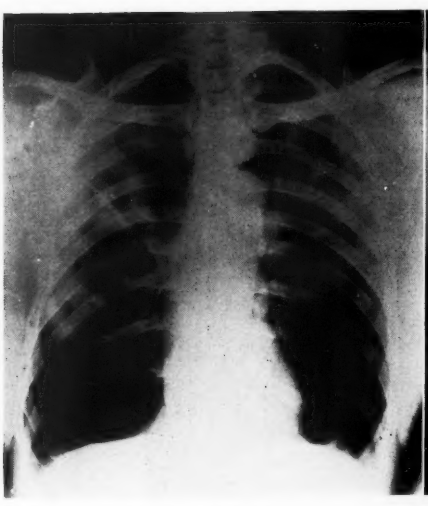


Fig. 16.

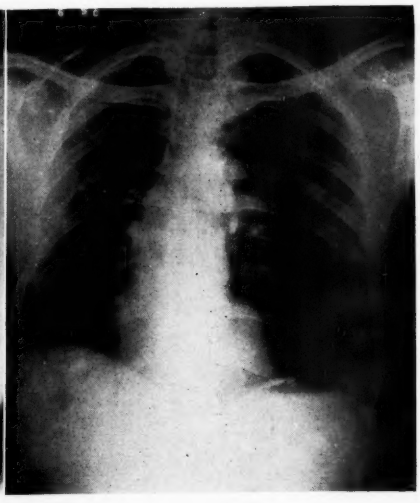


Fig. 17.

Fig. 16 (Case 7).—Chest radiograph showing bilateral basilar emphysematous cysts.

Fig. 17 (Case 7).—Radiographic appearance of chest following right thoracotomy with local excision of multiple blebs attached to upper, middle, and lower lobes. A second bleb is now visible on the left in the first and second anterior interspace laterally.

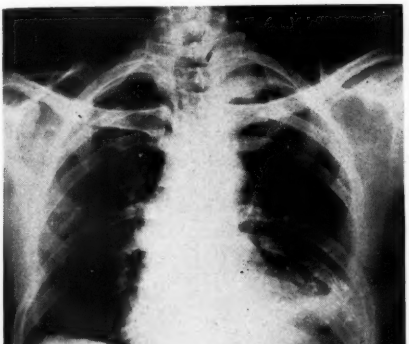


Fig 18 (Case 7).—Roentgenogram two weeks following second (left) thoracotomy with excision of multiple subpleural blebs left upper and lower lobes.

upper and lower lobes without sacrifice of functioning pulmonary tissue. His lung expanded promptly and he was discharged from the hospital on the tenth postoperative day. When seen one year later he had no chest complaints and had been in good health since discharge from the hospital.

CASE 9 (E. W.).—This 25-year-old man reported to the hospital in August, 1948, and stated that he had been unable to work since 1943 because of "chronic pneumothorax." He had been told that his condition could not be treated. Chest roentgenogram showed the apex of the right lung occupied by what appeared to be an emphysematous bleb with compression of the underlying lung practically identical with the roentgen appearance of Case 2. The remainder of the physical examination was normal. The patient's youth and apparent invalidism encouraged us to advise surgery and at operation the right upper lobe exclusive of the anterior segment was occupied by a large bulla. A segmental resection of the right upper lobe sparing the anterior segment was performed. Postoperatively his course was smooth except for a moderately severe blood transfusion reaction and an immediate atelectasis of the remaining right upper anterior segment which was adequately treated by tracheal suction. Convalescence has been entirely satisfactory, "thoracic" fear has disappeared, and the patient is able to carry out a normal existence.

CASE 10 (R. S.).—This 52-year-old man had been bothered with a cough for twenty-five years and was first seen by us in November, 1948. His cough was gradually becoming more productive and when first seen the sputum amounted to approximately three ounces per day. He had noted increasing dyspnea and fatigability for the past three years. On physical examination the positive findings were limited to the chest. Thoracic roentgenograms showed extremely dense bronchovascular markings in both bases with a marked degree of bullous emphysema occupying the left apex. Bronchograms demonstrated minimal bronchiectasis at the left base with crowding of all bronchi on the left suggesting compression by the bullous emphysema superiorly. Left thoracotomy was performed on Dec. 17, 1948, and a large emphysematous bleb was removed from the left upper lobe by local excision. The minimal bronchiectasis described in the bronchogram was not disturbed. He was ready for discharge from the hospital two weeks later when he suddenly experienced a sudden precordial pain extending down his left arm. Electrocardiographic examination showed a posterior coronary occlusion quite different from an entirely normal tracing obtained two weeks prior to surgery. This attack subsided with treatment but ultimately he succumbed to a second attack four months later. Post-mortem examination showed definite myocardial infarction. The left lung was fully expanded and adherent to the thoracic wall.

CASE 11 (B. T.).—This 35-year-old man was seen in July, 1949, because of exertional dyspnea which he experienced in his work as a warehouseman. He had been hospitalized on previous occasions for evaluation of bilateral pulmonary cysts in the upper lobes. His complaints were of two years' duration and chest roentgenogram showed bullous emphysema in the apices with the greater involvement on the right side. Bronchogram and bronchoscopy gave no additional information. On Sept. 12, 1949, three large emphysematous blebs were locally excised at their attachments to the right upper and lower lobes. There was some difficulty experienced in the expansion of the right lung due to a persistent bronchopleural fistula. Phrenemphraxis and pneumoperitoneum for a period of thirty days brought about complete expansion. The patient was last seen on April 2, 1950, seven months following surgery. He is greatly improved and is planning on left-sided resection in the near future.

CASE 12 (R. H.).—The reasons for this 50-year-old person's admission to the hospital were somewhat different from those of the previously described cases. He arrived via ambulance, was vomiting blood, had a severe secondary anemia, and was intoxicated. The patient was a chronic alcoholic and had had three gastric operations. Roentgenograms of the gastrointestinal tract showed a stomal ulcer at the site of previous resection of the

stomach and vagotomy was felt in order. Laparotomy was considered inadvisable and thoracic surgical consultation was requested. Chest roentgenogram revealed tremendous emphysematous blebs bilaterally distributed and occupying 50 per cent of each pleural cavity. Transthoracic vagotomy was performed on April 18, 1947, and six large emphysematous blebs were locally excised from the upper and lower lobes. Postoperatively the patient developed a moderately large pleural effusion which was adequately handled by needle aspiration and which likewise helped in the prevention of overdistention of the contralateral blebs. He was admitted to the hospital eight months after surgery for reexamination. The left lung was clear and there was no change on the right side. Barium studies of the gastrointestinal tract failed to show the previously described stomal ulcer.

CASE 13 (H. G.).—This 41-year-old Negro man had been in excellent general health until May, 1943, when he contracted a severe pneumonia. He was hospitalized and had a rather stormy course but was discharged in three weeks without apparent residual disease. Since then he suffered from frequent respiratory infections, the current one occurring during the hospital admission on Sept. 7, 1948. The main complaints were cough, dyspnea, and left-sided thoracic pain. Physical examination revealed a well-built, robust-appearing Negro in excellent physical condition. The only abnormal finding was a large area in the left apex interpreted as a bleb or localized pneumothorax. Bronchoscopy showed a marked distortion of the left upper lobe orifice; bronchography revealed failure of filling of the involved area, without associated bronchiectasis. On Oct. 29, 1948, left thoracotomy revealed a large subpleural bleb occupying the entire apex and compressing the upper lobe inferiorly. Removal of the bleb was accomplished by wedge resection of the adjoining lung with relatively little sacrifice of pulmonary tissue. The patient's convalescence was most uneventful and he has returned to a gainful occupation.

CASE 14 (M. D.).—This 31-year-old woman was admitted to the hospital on April 9, 1948, with a left spontaneous tension pneumothorax. As an emergency measure an intercostal tube was inserted and connected to underwater seal and the lung expanded within forty-eight hours. Roentgen examination of the chest revealed a generalized emphysema on the left with an emphysematous bleb occupying the lower portion of the right lung field. Six months later the patient was rehospitalized because of dyspnea, cyanosis, and exhaustion. On Nov. 29, 1948, she was again admitted to the hospital in critical condition. She weighed 85 pounds, was bedridden, orthopneic, and dyspneic with the slightest exertion. Roentgen examination revealed a marked progression of the right-sided bleb with mediastinal shift to the left and superior compression of the right lung. A right thoracotomy was performed on Dec. 7, 1948. Induction and initial maintenance of anesthesia were difficult but as soon as the chest was opened and the pressure relieved, the patient's condition immediately became satisfactorily stabilized. A large emphysematous bleb had replaced the entire right lower lobe. A right lower lobectomy was performed and several small blebs were excised from the upper lobe. The patient had a stormy first postoperative day due to retained bronchial secretions but bronchoscopy and supportive measures adequately handled this difficulty. She was discharged from the hospital one month later markedly improved. A minor complication was a small basal empyema which responded to adequate drainage.

SUMMARY

The chronological therapeutic approach to the problem of giant emphysematous blebs and pulmonary tension cysts has been presented and various surgical techniques hitherto recommended are given in detail.

In the majority of cases, large emphysematous tension cysts are progressive and lethal in infants and surgical excision offers the best therapy for survival. The adult having a minimum of symptoms or who has no chest complaints whatsoever, but does present the roentgen appearance of localized

emphysematous blebs, should be given the opportunity of benign thoracotomy before the possible development of life endangering tension or intracavitary suppuration. In bilateral cases, the patient's condition at operation improves as soon as the chest is opened, however, preoperative provision should be made for the inevitable postoperative distention of the contralateral bleb or blebs.

Fourteen patients having giant emphysematous blebs and pulmonary tension cysts are presented. A total of fifteen operations was performed in the series and comprised ten local excisions, three segmental resections, and two total lobectomies. Six of the cases had bilateral disease, one of these being subjected to staged thoracotomy on either side with complete removal of all visible disease. There is definite improvement in twelve of the fourteen cases without serious operative morbidity. There was one operative death, this in an elderly man with bilateral disease, and it was due to overdistention of the contralateral bleb. This mortality could probably have been avoided by preoperative decompression of the opposite side. The second death occurred four months following surgery and was due to autopsy-confirmed coronary occlusion.

Postoperative complications included an osteomyelitis of a resected rib (Case 1), a persistent bronchopleural fistula (Case 11), and a localized empyema (Case 14). All of these situations responded promptly to therapy.

While the postoperative observation in these patients is not sufficiently long to warrant conclusive opinions, the preliminary results in this series of cases are felt worthy of presentation.

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2938 MCCLURE ST.

DISCUSSION

DR. R. K. HOLLINGSWORTH, Martinsburg, W. Va.—I have been very much interested in the subject of this presentation. Much confusion exists in the classification of pulmonary cysts, blebs, and bullae, and they do present a real challenge. We are seeing an increasing number of cases of this type of pulmonary disease, more in the older age group. We have been doing bronchospirometric studies and feel that they are of great importance, and I would like to present two cases in illustration of this.

(Slide.) The first patient is a young man of about 28, who was sent in following his second episode of spontaneous pneumothorax; you can see these large blebs in the apex on the right. He had no symptoms, but he was a farmer, worked hard, and we felt that something should be done about it.

(Slide.) In the record of bronchospirometry, we see that the ventiliation, vital capacity, ant oxygen consumption are about normal, perhaps even favor the right side; he has 60 per cent oxygen consumption on the right. We felt, though, that in view of the history of spontaneous pneumothorax on two occasions, we should proceed with operation, and we resected a bleb, a large bleb; no lung tissue was involved and no lung tissue was removed.

(Slide.) This is the postoperative film, and we hope that the apex will stick there and that he will have no further trouble.

(Slide.) This was a 55-year-old man who came in because of dyspnea; you see large bullae on the left side. There is nothing seen on the right, but we got some films made three years previously (slide) and the ones on the left were small at that time. There was quite a large lesion in the right upper lobe. He had no treatment, but some time in the two years from 1947 to 1949, this air cyst (right side) disappeared, while the one on the left side became increasingly larger and under great tension.

(Slide.) Here are the bronchospirometric studies; on the right side he has 72 per cent oxygen uptake and only 28 per cent on the left. Because of his age and the fact that he had had alesion on the right side, we felt that we would rather try conservative treatment. We therefore instituted drainage by the method as presented by Dr. Head. Just before I left home we have a second spirometric study and this figure (vital capacity) was right, 1,200; left, 900—but the oxygen consumption was equal on the two sides, 190 c.c. right and 190 c.c. left, making it 50/50.

(Slide.) He has good expansion here, although there is still a very small area high in the apex.

DR. LEO ELOSSER, San Francisco.—I wonder whether we should not pay just a little more attention to the varying pathology concerned in these various forms of peripheral dilatation in the lung. It seems to me that there are various diseases that are sometimes confounded. The solitary pulmonary cyst, congenital in origin, perhaps due to some anomaly of bronchial or bronchiolar development, is one thing. Diffuse cystic disease of the lung, congenital, is another thing. Emphysema, with bullae, acquired for one reason or another in the course of a man's life, is still a third thing. It seems to me that the pathology is quite important with regard to methods of treatment. I wonder, perhaps, whether Dr. Dugan's first case in an infant was not a solitary cyst, and whether another one of the cases was not also a solitary cyst. I am quite sure that Dr. Robert Miller's case, which Dr. Dugan mentioned, was a solitary cyst. The pathology is quite different in those infants with congenital disease of the lung, from what it is in acquired emphysema that has arisen in the course of years in older men.

DR. M. D. TYSON, Hanover, N. H.—In 1936, I operated upon a patient with an emphysematous bleb causing recurrent pneumothorax, and Dr. Sycamore reported it in that year. Dr. Crandell and I reported it later. From our perusal of the literature, we thought it was the first bleb to be diagnosed, and a planned operation carried out for the cure of spontaneous pneumothorax. This impression was borne out in a paper by Dr. Dolley and his associates published this year.

(Slide.) This 30-year-old man came in with his fourth attack of spontaneous pneumothorax; he was in extremis, with cyanosis, orthopnia, and rapid pulse. After numerous operations, the lung re-expanded somewhat and (slide) this slide shows the outline of a bleb at the upper pole. He refused treatment at that time and went out, but came back again in four days with a recurrence, his fifth attack of spontaneous pneumothorax. At this time he was more amenable to suggestion, and operation was carried out with removal of the bleb.

Since the operation was done in 1936, I thought it would be timely to give a follow-up on this case. He went along well for five or six years; never had any further pneumothorax, but had gradual development of symptoms from emphysema, which resulted in numerous attacks of heart failure. He developed cor pulmonale, and died in 1947, eleven years after the operation.

From this and subsequent cases, it is our feeling that, though operations for removal of emphysematous blebs and cysts are often worth while, these cases frequently have a shortened life expectancy due to the underlying generalized emphysema.

DR. E. E. AVERY, Chicago.—Little is known regarding the pathogenesis of emphysematous blebs and bullae and we readily admit our abysmal ignorance in this subject. Dr. Head and I have been working on this study since 1945, when several cases came to our attention. These patients were extremely dyspneic and bedfast with far-advanced bullous emphysema. It was felt that they would tolerate nothing but the mildest type of surgical therapy, therefore catheter suction drainage was tried. Somewhat to our surprise, and certainly to our gratification, it worked very well. We have been seeing an increasing number of these cases and it seems likely that this will become a more common disease entity because, with the antibiotics, patients are surviving severe respiratory diseases and more people are living into the older decades in which emphysema is more prevalent. Emphysematous bullae should be suspected and sought in all patients with gradually increasing dyspnea, spontaneous pneumothorax, or with a history of asthma.

I noticed that many of the films shown today were overexposed x-ray pictures. It is important to cut down the kilovolt or the milliampere seconds from the normal calculated for the patient and take these films at a much lighter penetration so as not to burn out the areas that will give the diagnosis. Films should be taken in many views; obliques, laterals, lordotic, etc. We have found it helpful to have the patient cough forcefully, and then take inspiratory and expiratory films with Valsalva technic. In this manner air is forced into the cystic areas, causing more compression of the surrounding lung and increased visibility on the x-ray plate. We are now routinely using laminagraphy on these patients. It is useful in determining the number, and the exact location, of the cavities for the exact positioning of catheters. Bronchography is used and is also helpful in determining the location of the dilated and the compressed segments.

Preoperatively, it is often difficult to determine exactly whether one is dealing with a bleb, a bulla, or a congenital cyst. If we feel that the patient might benefit from suction drainage, an extrapleural gauze pack is inserted to produce adhesions, and is removed in two weeks. A trocar and thoracoscope are introduced for inspection of the cavity. A diagnosis can almost always be accurately made from this examination and the future treatment anticipated. The interior of a bleb presents a spherical cavity with thin, translucent, whitish wall, with very few pinpoint alveolar communications on the parenchymal side. In a bulla, you see a large area of grossly dilated, stretched, and shredded lung tissues giving a cobweblike appearance of the base of the cavity. A congenital cyst has a heavier wall. The cavity, which is lined with epithelium, separates segments of lung

causing an indentation of adjacent segmental bronchi and vessels within the wall of the cyst. Air fistulas are seen along the course of the segmental bronchi. Resection is the treatment of choice when congenital cysts are found.

In summary, then, we like to use catheter suction drainage in large, unilocular, solitary blebs and bullae with or without incapacitating dyspnea. On occasion it is useful in multiple bullae where the patient has severe dyspnea and is in poor condition. By decompressing the larger bullae, we have been pleased to see many of these patients return to functional capacity.

DR. JEROME HEAD, Chicago.—I feel that the surgical relief of dyspnea is one of the new fields in thoracic surgery, and that it has a much larger future than anyone expects at the present time. I feel also that it is much more complicated than anyone expects and that the pathology which causes dyspnea and which can be relieved by surgery is not single and simple but is multiple and complicated. At the present time our surgical procedures are limited to the removal of large cysts and of emphysematous blebs and bullae which are compressing the lung. I feel certain that not infrequently pulmonary lobes and pulmonary segments become markedly emphysematous and crowd adjacent normal lung tissue in the same manner that it is crowded by bullae and blebs. It seems quite probable, therefore, that in the future a careful study of cases and a careful selection of cases will reveal some in which resection of such emphysematous segments or lobes will relieve serious respiratory insufficiency.

I agree with Dr. Eloesser that the first case which Dr. Dugan presented was probably a congenital cyst of the lung and not an emphysematous bleb. We have recently seen a premature infant who breathed poorly after birth. The roentgenogram taken at that time showed a small cyst in the right middle lobe. The respiratory difficulty became greater and the roentgenogram taken a week later showed that the cyst occupied half of the thorax. A roentgenogram taken a week later showed that it occupied the entire thorax. The lung had entirely disappeared. Shortly after this the child died.

When one sees a ballooning cyst or bleb in the thorax, he ordinarily thinks that this ballooning is due to a positive pressure within. This is not necessarily so. I am sure that they balloon for an entirely different reason. When one puts a catheter or a needle into a large cyst and measures the pressure, one finds that the pressure is plus and minus zero; in other words, that there is not a positive pressure in the cyst. If one considers the mechanism carefully, he will conclude that any sac within the thorax which is stretchable but not elastic will gradually stretch until it takes over as much of the thorax as it can stretch to fill. These cysts, then, are not tension cysts but are stretched cysts which, being inelastic, stretch more and more with each inspiratory effort, and, because they are inelastic and therefore fail to retract, grow steadily larger.

DR. OSLER A. ABBOTT, Atlanta.—I rise mainly to repeat our feeling that there is a role of autonomic neurosurgery in conjunction with the treatment of such patients. We find it is very seldom that we see emphysematous blebs of major size which are not associated with rather extensive and diffuse interstitial emphysema. We feel that there is a definite place for the laminagram, inasmuch as it shows up the bronchovascular pattern, demonstrating to us compressed lung tissue which we could not otherwise find in a patient who is completely debilitated and for whom a rather drastic procedure may be justified. The patients we have done, which number nineteen, who have had autonomic surgery in relation to the emphysema, have included some patients in whom, on a maximum breathing capacity test, we practically had to assist them to walk down the hall. Some of them had been completely confined to oxygen tents. Very dramatic results have been obtained. They have shown a definite response to bronchial-dilating drugs before being operated upon. I would like to show three slides to demonstrate some of the factors in the initial evaluation of these patients.

(Slide.) This is a patient with an expiration and inspiration film, in which one might feel there were major blebs, certainly as indicated by the slide on the right, with multiple

corrugations over the diaphragm which disappear on expiration; there was shifting of the heart to the contralateral side during expiration with the diaphragm actually descending on expiration. There is a very small bleb here, but this patient's major difficulty was interstitial emphysema and a plication procedure to relieve the size of the right lower lobe, plus denervation procedures, rehabilitated this patient.

We frequently note a change in collapsibility of an emphysematous lung following denervation; the lung was not collapsed when first entering the thorax; it will collapse on denervation.

(Slide.) I wish to show one slide to emphasize imitation of pulmonary fibrosis by interstitial emphysema. This type of patient one might say has extreme pulmonary fibrosis. The granular markings on these films are not due to fibrosis; at surgery it was shown to be an indication of extensive interstitial emphysema. This patient has been completely rehabilitated and is working full time as a mechanic, and has multiplied his maximum breathing capacity four times following operation. Therefore, I feel it is important to stress the adjunct of nerve surgery in these cases, and the interpretation of the fibrosis as being truly interstitial emphysema in many instances.

DR. LAWRENCE MISCALL, New York.—I would like to show three slides illustrating the use of angiocardiography in the preoperative evaluation of how much functional pulmonary tissue is available for re-expansion after excision of such cystic areas.

(Slide.) This is the regular flat plate, in an individual who had severe dyspnea due to bullous disease which involved the major portion of both upper lung fields.

(Slide.) Here is the visualization of the vascular tree. On both sides no upper lobe branches can be seen. The vascular pattern that can be seen is displaced inferiorly and adjacent to the heart. It conforms to the pattern of the lower lobes.

(Slide.) This individual had a left upper lobectomy for cystic destruction of the entire lobe. Nothing has been done on the right side. I think you can see in this slide the marked change in the vascular pattern on the left side which has followed the expansion of the lower lobe to about normal size. The pleural tent has been used to prevent overdistention. The right side has not been operated upon. These angiocardiographs gave fairly accurate information concerning the functional state of the lung. The area not visualized was completely cystic while the area that would re-expand was well visualized.

DR. SAMSON (Closing).—Dr. Dugan and I wish to thank the discussors for their interesting observations. Time does not permit individual answering of all the questions raised, but if those who wish to have that sort of service will meet us outside we will be glad to do what we can after the meeting.

Certainly Dr. Hollingsworth, Dr. Miscall, and Dr. Abbott have given us additional means by which preoperative investigation can be made, and in many cases this is advisable. We have not done them primarily, we must admit, because facilities have not been at hand.

In response to Dr. Eloesser, we agree that the estimation of the pathology at surgery is of utmost importance. We still insist on our prerogative, however, having seen Case 1 at surgery, and a similar case only a month or so older—we insist that these were emphysematous blebs, solitary to be sure, but still typical subpleural collections of air with scrabbled-out lung tissue at the base. The second case, incidentally, showed a considerable amount of emphysema in the upper lobe. I think it is important to realize that in many instances we are dealing with generalized disease. We admit that many of these patients will probably die of the generalized disease, namely, emphysema, at some future time. An appreciable percentage, however, show no gross evidence at least, of emphysema in the lungs where single or several cysts are removed.

In so far as Dr. Head is concerned, he promised us a little "what for" several days ago, and I am sorry that our estimable President cut him off before he had a chance to complete his critique. At any rate, I think this presentation merely shows that there are several ways of skinning a cat. We believe, particularly in the case of multiple cystic

areas, that excisional therapy is probably to be preferred. We realize that tube drainage such as Dr. Head and Dr. Avery demonstrated two years ago and brought up to date today, certainly has its place if we can prove that these blebs are single and not multiple.

It seems unfortunate that Dr. Head's premature infant did not have the benefit of surgical intervention for an obviously progressive and lethal lesion.

I must confess that I am confused at Dr. Head's attempted differentiation between tension and pressure cysts and "stretch cysts." Whether or not the pressures are greater than atmospheric, the cysts are space-occupying, and cause collapse of parenchyma and mediastinal shift, which amounts to the same thing so far as I am concerned. Comparison of our Figs. 11 and 12 is a case in point. Preoperatively, the trachea is displaced to the left, whether from retraction on the left or pressure on the right could not be ascertained. Postoperatively, the giant bleb having been removed, the trachea has returned to the midline.

TREATMENT OF GIANT CYSTS OF THE LUNG

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THE giant cyst in the lung is an enigmatic lesion. Its etiology is usually obscure, its behavior puzzling, and the diagnosis is frequently difficult. Its presence may be indicated only by the onset of a complication of the lesion, or, on the other hand, its size alone may be sufficient to produce disabling symptoms. Attempts to classify cysts of the lung often add to the confusion and the histologic classification may vary from the clinical classification.

A classification based upon congenital or acquired origin with subdivisions dependent upon the histologic character of lining membrane and the size and number of cysts present, will clarify the type of disease under consideration. Changes of pressure in the bronchi or thorax will greatly influence air-containing cystic lesions within the lung. The thin wall may be covered by a bronchial type of epithelium and fragments of cartilage, muscle, and elastic fibers may be found in the wall; however, both lining membrane and wall may be composed only of compressed alveoli and connective tissue. Different portions of the large cyst walls may have different types of lining tissue, depending upon the presence of infection or hemorrhage and the rate of expansion of the cyst. Cysts arising in aberrant lung tissue that do not develop a bronchial communication represent true congenital cysts for they have developed within pulmonary tissue without function and without communication with the trachea or its branches. The uncomplicated cyst that develops in a grossly emphysematous lung represents an acquired cyst for it is related directly to the usual inflation and deflation of the lung that occurs in the functioning lung. The size and number may vary from the single giant cysts under consideration to the "frothy" lung that is entirely composed of small cystic areas.

PATHOGENESIS

Miller[13, 14] believes both blebs and bullae are acquired localized air pockets within the lung, differing in location. Blebs are formed by rupture of an alveolus directly beneath the pleura with the escape of air into the areolar layer of the pleura. The escaping air extends through this areolar layer separating it from the underlying alveoli. The extension may be arrested by septa demarcating a secondary lobule, or it may extend beyond the confines of one lobule. In contradistinction to a bleb, a bulla results from rupture of an alveolus into adjoining alveoli rather than by dissection of the pleura from the underlying alveoli. There is a continued accumulation of air so that, eventually, a large air space may form. The pleura retains its connection with the subjacent alveoli but may be tightly stretched over the expanding bulla. The alveoli bordering the bulla are compressed. The lining of such cavities is composed of fibrous tissue and alveolar epithelium.

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The single bulla or bleb is closely related to the positive pressure cavities secondary to infection, or the cysts seen in emphysema. Factors common to all are (1) bronchiolar or alveolar damage, (2) fibrous tissue formation, and (3) a free ingress of air into the space, with an impeded egress.[16] This latter factor results from the tortuosity of the respiratory bronchioles feeding the space, a flaplike valve action of tissue surrounding the opening, or edema of the mucosa of the bronchiole entering the space. In the latter instance the widening of the bronchiole during inspiration allows air to enter but its narrowed diameter during expiration may be sufficient to prevent the escape of the air. On the other hand, from thoracoscopic observation of the interior of large cysts Head and Avery believe that inflation of the cyst occurs during the expiratory phase of respiration. As intrathoracic pressure increases during expiration the pressure within the lung encapsulating the cyst becomes greater than that within the cyst, and air from the lung parenchyma is forced into the space. The surrounding parenchyma bulges into the cavity. With inspiration the intrathoracic pressure decreases and the air space expands because the pressure is greater than in the encapsulating lung, which is then compressed and the minute bronchiolar openings are occluded. The air is then trapped within the cavity.[8]

Coughing, straining, and crying accelerates the process just described. From direct observation of the interior of the cyst with the thorax opened, it has seemed to us that the bronchioles were open during the inspiratory phase of respiration and closed during the expiratory phase, the tissue near the orifice acting as a simple flap valve. As would be expected from their origin large bullae and blebs may not differ greatly in appearance grossly or microscopically. The bulla may reach sufficient size to compress markedly the subpleural alveoli and appear to have an external surface of pleura alone; however, this external layer is thicker, more fibrous, and bleeds considerably more when incised than the external surface of a simple bleb. The bulla frequently reaches a greater size than the bleb and the bronchiolar openings are greater in number. The bleb is more prone to spontaneous rupture than is the bulla and frequently accounts for spontaneous pneumothorax.[2, 10, 12]

Cysts of equal size and the same clinical characteristics may also arise from a congenital lesion. The space must either be collapsed at the time of birth, or be filled with fluid. In the latter instance, the fluid must be evacuated before the cyst can be filled with air. Such an event was suggested by the case report of Anspach and Wolman.[1] It hardly seems likely that a cyst of small size could reach the tremendous proportions reported,[4, 7, 18] within a few days or weeks after birth, and still retain an intact epithelial lining if only ballooning of a small cyst had occurred; therefore, it is probable that cysts were present at the time of birth and subsequently enlarged to the extent that symptoms were produced. There are other cysts of congenital origin that may be of small size, multiple in number, and asymptomatic until later in life. The congenital cysts are lined by bronchial epithelium and smooth muscle and cartilage may be found within the cyst wall. While this type of lining is

usually accepted as distinguishing a congenital cyst, it is the opinion of some that cysts having similar structure can be acquired.[15] There is one characteristic of single large cysts of either type that has a bearing upon the plan of treatment. It is not unusual to find the entire hemithorax occupied by the lesion; the lobe or lung appears to be damaged beyond repair and the initial impression is that it should be sacrificed. Yet when the cyst wall is incised and the pressure reduced, the surrounding lung parenchyma promptly expands to fill the pleural space. Preceding the opening of the cyst, it may appear that several cysts are present on the surface of the lung, while in reality, the cystic areas are only loculations which communicate freely with the main body of the space and are separated only by attenuated lung parenchyma from adjoining loculations. The base of the cyst is trabeculated and usually several bronchiolar orifices are evident. The extent of the cyst may be such that it seems to arise from the hilum while, in reality, the hilar structures are only displaced by the lesion. When the base of the cyst is exposed it is usually surprisingly small and continues to diminish in relative size as the compressed portions of lung regain their normal configuration. It becomes evident that the amount of lung tissue that must be sacrificed is small and only includes (1) those portions of the parenchyma immediately adjacent to the cyst, (2) the portions so attenuated between loculations that the viability may be compromised when the lining tissue is removed, and (3) that area at the base required for secure closure of the bronchiolar orifices.

DIAGNOSIS

The symptoms resulting from the presence of single large cystic lesions are dependent upon the size of the lesion and the complications superimposed by infection, hemorrhage, and spontaneous pneumothorax. The lesion increases in size at the expense of the surrounding tissue and consequently may cause symptoms indistinguishable from those of a tension pneumothorax.

Infection is uncommon in blebs and bullae but is common in congenital cystic disease and is often the presenting symptom. Hemorrhage also is more usual in diffuse cystic disease than in single large cystic lesions. Spontaneous pneumothorax is a common complication of blebs and bullae and they, in turn, are the common etiologic factor in spontaneous pneumothorax in nontuberculous patients.[2] The findings on examination of the chest again depend on the size of the lesion and the presence of a complication. At times the physical findings are indistinguishable from those of pneumothorax, which may also coexist. In the presence of infection widespread pneumonitis may be present. An increase in size of the hemithorax on the involved side is often found, with widening of the interspaces and a decrease in the respiratory movements.

Roentgenographically, the cyst appears as an area of decreased density with a fine border, in contrast to the dense border often seen in inflammatory lesions. A diffuse haziness about the annular border indicates areas of compression of the adjacent parenchyma. Fine linear shadows may cross the area of decreased density to indicate trabeculae within the cystic area. A cyst of large size may be misinterpreted roentgenographically as a tension pneu-

mothorax. However, there are generally some differential points which too often are found only after reviewing the roentgenograms. In the presence of a pneumothorax the elastic lung will generally contract about the hilum and will appear as a globular shadow while with a "vanishing lung" the hilar shadow is generally elongated and areas of compressed lung may be seen in the apex of the thorax or over the diaphragm. The widening of the intercostal spaces produced by large cysts may be apparent (Fig. 3), as opposed to the narrowing of the interspaces that is found in chronic pneumothorax.[9]

TREATMENT

The therapy of cystic lesions has been varied. Treatment based on external compression of the lesion has failed.[5] External drainage has only been partially successful. In reviewing the literature it seems evident that some lesions responded well to external drainage by intracavitary suction (Monaldi),[8] or by an endocutaneous flap using Eloesser's method.[3] However, such methods are not always effective as indicated by the reports of prolonged open drainage of cysts without spontaneous closure.[11] The lesion has often been mistaken for thoracic empyema preoperatively and generally these cysts have required open drainage.

It is paradoxical that lesions appearing to be of similar nature respond differently to similar treatment. It seems likely that a different pathogenesis exists, although the lesions seem to be clinically identical. It is possible that the difference lies in the character of the lining membrane; those cysts lined by respiratory epithelium resisting closure, and those lined by alveolar and fibrous tissue closing promptly when the balloon effect is eliminated and the walls are apposed. Throughout the case reports in the literature there is consistent difficulty in distinguishing the cysts lined by epithelium from those without epithelial lining before histologic study of the tissue. Excision of the lobe or lung containing the cyst has been effective in treatment[4, 7, 15, 18, 19] but has the objection of sacrificing nondiseased lung tissue in many instances. Closure of the fistula followed by suture of the opposing walls of the cyst has recently been advocated by Naclerio and Langer.[17] This method is advantageous in sacrificing the minimal amount of lung tissue, but in cysts lined by epithelium the opposed surfaces might fail to unite. Chemical cauterization of the surface of the cyst in order to sclerose the wall and close the bronchiolar orifices has been used successfully[6] but is an unpredictable method.

In view of our inability to differentiate between epithelialized and non-epithelialized balloon cysts in the lung, we have directed our treatment toward excising the wall of the cyst, closing the bronchiolar orifices, sacrificing only that lung parenchyma adjacent to the cyst, and re-expanding the lung by temporary suction drainage postoperatively. The involved lobe or lung has not been removed in any instance, since it seemed a needless sacrifice of normally functioning pulmonary tissue. In those instances of multiple bilateral large cysts, as in the cases cited by Head and Avery[8] intracavitary suction drainage may be indicated rather than thoracotomy and excision of the cyst wall.

CASE REPORTS

CASE 1.—A 25-year-old white woman had been free of symptoms until six weeks preceding admission to the hospital. At that time she had been seen by her physician for treatment of an upper respiratory infection. She had no other respiratory symptoms but had persistently been below her expected weight and had chronic fatigue. There was no history of dyspnea. There were signs of a left tension pneumothorax. She was observed at weekly intervals and as there was no evidence of re-expansion of the lung she was referred to this hospital for treatment.

The patient appeared chronically ill, weight was 95 pounds. The significant physical findings were those of a left tension pneumothroax (Fig. 1, *A*). A left thoracentesis was done; 5,000 c.c. of air were removed without change of pressure or expansion of the lung. Following this, air escaped into the pleural space and delineated the cyst margin, (Fig. 1, *B*). On May 17, 1948, a left thoracotomy was performed and a large cyst found which filled the hemithorax. It arose from the anterior segment of the left upper lobe. When the cyst was opened it promptly deflated and multiple small bronchiolar orifices were evident in the trabeculated base. The surrounding lung parenchyma promptly inflated. The cyst was excised and suction drainage of the pleural space was established.

The patient had an uneventful postoperative course with re-expansion of the lung, and was discharged from the hospital on the fourteenth postoperative day. Microscopic examination of the excised cyst wall showed only connective tissue. Six months after operation the patient had gained twenty pounds, was asymptomatic, and the lung was well expanded (Fig. 1, *C*). She had no fatigue with normal activity.

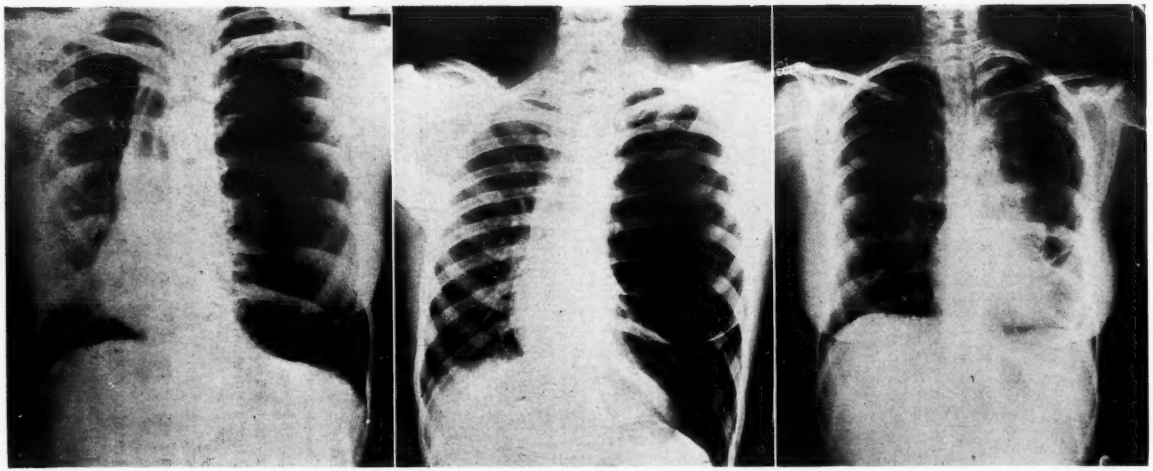
Comment.—It is hardly conceivable that the cyst would reach a size sufficient to compress both the homolateral lung and ipsilateral lung without giving severe dyspnea. A rapid reduction of lung volume to this extent would certainly produce symptoms. One can only speculate on the likelihood of chronic oxygen want preceding obliteration of the lesion due to the gross diminution of functioning pulmonary tissue. Postoperatively the loss of fatigue, the gain in weight, and general sense of great improvement would suggest the possibility of the correction of a chronic anoxia.

CASE 2.—A 16-year-old white boy had been free of symptoms until three years preceding admission, when he first noticed shortness of breath with exercise. One year preceding admission dyspnea had been particularly marked when playing basketball. Six months later a survey chest roentgenogram was interpreted as showing a left pneumothorax. This had not changed significantly during the six months before admission to the hospital. Studies for the presence of tuberculosis were negative.

The only significant findings on admission were limited to the left hemithorax. There was widening of the interspaces on the left with increase in the size of the entire hemithorax. The findings of a large pneumothorax were present on the left, the mediastinum being displaced to the right (Fig. 2, *A* and *B*).

A left thoracentesis was performed and a gas specimen obtained from the cyst, which contained 6 per cent carbon dioxide and 8.9 per cent oxygen; the intracavitary pressure was +4 to −4 cm. of water.

On Dec. 10, 1948, thoracotomy was performed and a large cyst was found to occupy the left hemithorax. After opening the cyst two bronchiolar orifices could be seen at the trabeculated base. The origin of the cyst was from the left upper lobe. The bronchiolar openings were closed, the roof removed from the cyst, but the portion adherent to the lung parenchyma was left intact. The upper lobe promptly expanded. Adhesions binding the lower lobe inferiorly and medially were divided, which was followed by prompt expansion of the entire lung, although it did not completely fill the hemithorax. The chest was closed about a catheter for suction drainage. Histologic study of the excised cyst wall showed only connective tissue.

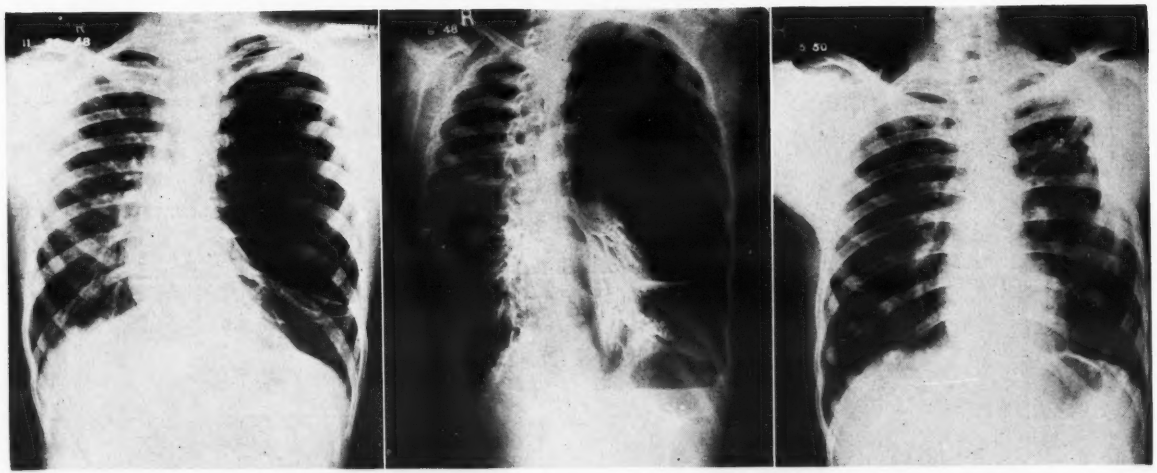


A. *B.* *C.*

Fig. 1.—*A*, Roentgenogram of Case 1 showing what appears to be a left pneumothorax with displacement of the heart and mediastinum into the right hemithorax.

B, Roentgenogram of Case 1 after aspiration of cyst. Gas had escaped into left pleural space and the thin cyst wall is delineated inferiorly. The collapsed lower lobe is now visible.

C, Six months postoperatively there is moderate pleural thickening in the left hemithorax with mediastinal shift to the left.



A. *B.* *C.*

Fig. 2.—*A*, Roentgenogram of Case 2. Note widening of interspaces of left hemithorax and compression of lung medially. *B*, Bronchogram of Case 2. Note crowding of all bronchial subdivisions. An artificial pneumothorax outlines the cyst wall. *C*, Roentgenogram one year postoperatively. The widening of the interspace on the left has persisted.

Postoperatively a small apical pneumothorax persisted. The drainage tube was removed on the fifth postoperative day. Development of an apical empyema required open drainage on the thirtieth postoperative day. The cavity was then spontaneously obliterated and the patient was discharged fifty-seven days after operation. He was free of symptoms one year later (Fig. 2, *C*).

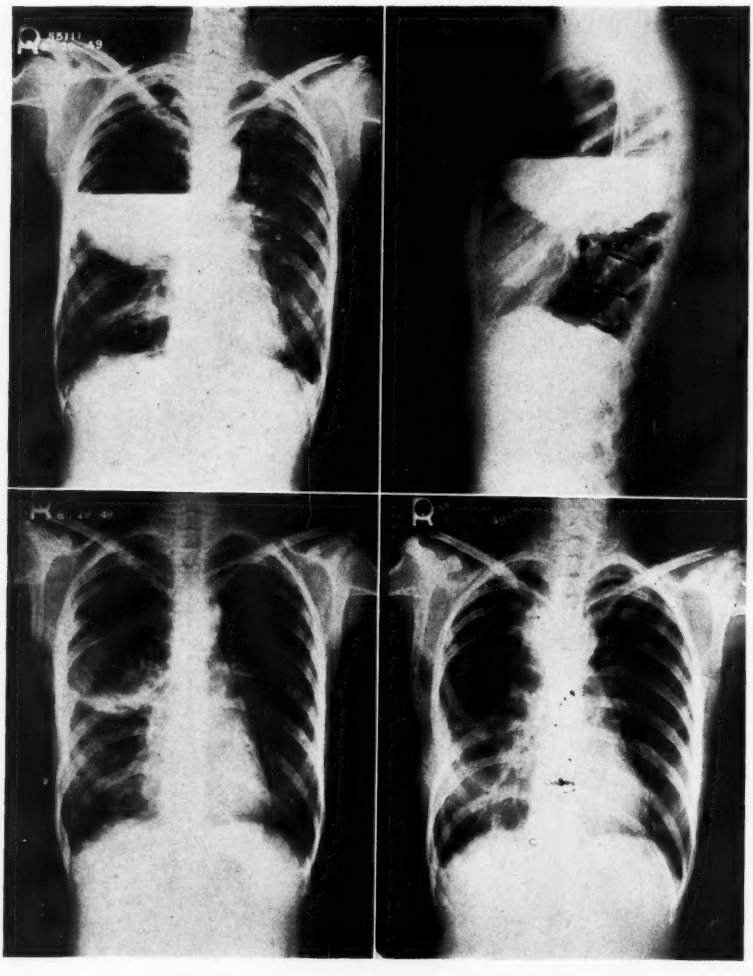


Fig. 3.—*A*, Anteroposterior roentgenogram of Case 3 on admission.

B, Lateral roentgenogram of Case 3 on admission.

C, Roentgenogram of Case 3 nine weeks following adequate dependent drainage. The size of the space has not decreased appreciably.

D, Roentgenogram of Case 3 four weeks following excision of the cyst. A small apical pneumothorax persisted but subsequently the lung re-expanded spontaneously.

Comment.—The presence of dyspnea on activity over a period of years suggests that this cyst had been present for a long time. The persistence of an apical pneumothorax followed by an infection of the space postoperatively indicates that a bronchiolar orifice was left unclosed at operation. In this instance a portion of the lining of the cyst wall was left intact which may have influenced the occurrence of infection.

CASE 3.—A 33-year-old white woman was admitted to the hospital with a draining sinus on the posterior surface of the right hemithorax. A thoracotomy had been done in this hospital June 23, 1949, and a large collection of pus was drained from the right hemithorax, with a diagnosis of infected giant lung cyst (Fig. 3, *A* and *B*). After discharge the large cavity had not decreased in size and a bronchopleural fistula had remained patent, with persistent profuse drainage.

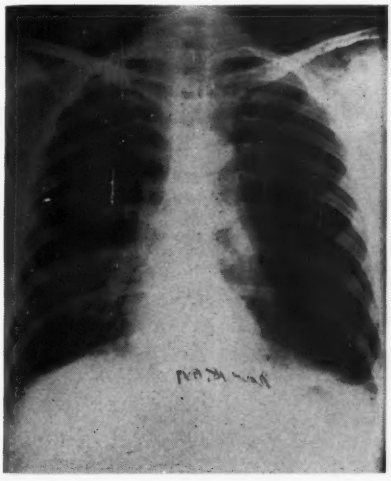


Fig. 4.—Roentgenogram of Case 4 on admission. There is a left pneumothorax of moderate size. The collapsed lower lobe is seen as a hilar mass, and upper lobe lung markings are visible.

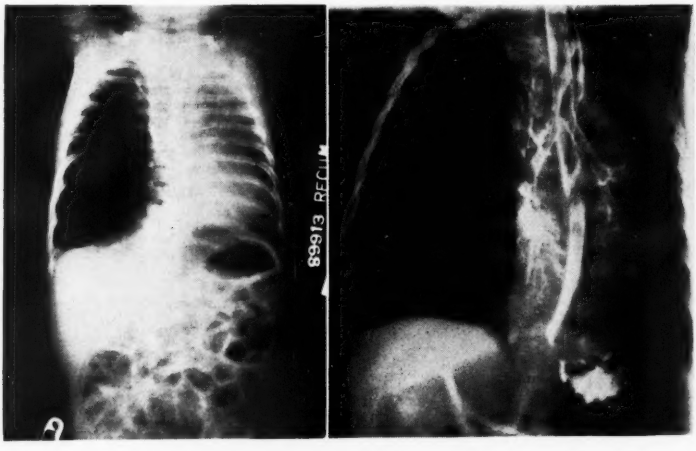
On the second admission the significant findings were limited to the right hemithorax. There was a granulating drainage tract through the thoracic wall at the area of the angle of the eighth rib. The tract entered a spacious cavity that appeared to be lined with epithelium (Fig. 3, *C*). The depth of the cavity was trabeculated and several fistulas were evident. There was a copious amount of thick pus within the cavity.

On Aug. 17, 1949, the sinus tract was widely opened to afford access to the cavity for cleansing; after a few days of treatment the secretion was greatly reduced. On Sept. 9, 1949, a thoracotomy was performed. The sinus was excised. A large cystic space was found filling the entire superior portion of the right pleural cavity. The space was opened widely, the wall of the cyst was excised, and the base was found to arise from the posterior segment of the right upper lobe. The base was excised and the bronchiolar orifices closed. The adhesions of the lung were then divided and the lung expanded promptly to fill the entire pleural space except for a small area at the apex. Catheter drainage of the apical area of the space was established and the wound closed by using a muscle flap from the trapezius and latissimus dorsi to close the defect incurred in the chest wall produced by extension of the fistulous tract. Histologic study of the cyst wall showed bronchiolar epithelium.

The postoperative course was uneventful and the patient was discharged on the seventeenth postoperative day with the wound well healed (Fig. 3, *D*). In a follow-up examination three months following operation she was free of symptoms; the lung was normally expanded.

Comment.—This large cyst was drained externally even though recognized as an infected giant cyst of the lung preoperatively because of concomitant pneumonitis. After drainage there was no tendency for the space to obliterate. Excision of the lining membrane of the cyst with closure of the bronchiolar orifices was required before the lung would re-expand. Only a small amount of lung tissue was sacrificed.

CASE 4.—A 61-year-old white man had a sudden attack of severe dyspnea three months preceding admission. He had an upper respiratory infection with paroxysms of coughing preceding the onset of dyspnea. His local physician found a left pneumothorax. Repeated aspirations of the chest and prolonged bed rest did not influence the collapsed lung. He was referred for hospital treatment because of the persistence of severe dyspnea and a left pneumothorax.



A. *B.*

Fig. 5.—*A*, Roentgenogram of Case 5 on admission. The heart is displaced to the left. The right lung appears to be compressed.

B, Bronchogram of Case 5. Note compression of the entire right lung along the mediastinum with shift of all mediastinal structures to the left.

The findings of significance were those of a left pneumothorax with complete collapse of the left lower lobe and partial expansion of the left upper lobe which was suspended from the apical thorax by adhesions (Fig. 4). Studies for tuberculosis were negative.

Thoracotomy was performed on Dec. 23, 1949. A large cyst arose from the anterior segment of the left upper lobe. Unfortunately the cyst was entered as the parietal pleura was incised and no definite point of spontaneous leakage was determined. A small trabeculated base with multiple bronchiolar orifices was found in the anterior segment of the left upper lobe. When the entire wall was excised and the orifices closed the entire lung promptly re-expanded. The chest was closed about a catheter drain for suction. Histologic study of the wall showed only connective and granulation tissue. The patient had an uneventful postoperative course, the lung expanded, and he was discharged from the hospital on the eighth postoperative day. He has been free of symptoms since discharge.

Comment.—This illustrates a common complication of blebs and bullae. It is surprising that clinical evidence of infection was not superimposed in view of the prolonged leakage through the cyst wall. The absence of any symptoms preceding an upper respiratory infection suggests that this cystic lesion may have been acquired quickly and was of short duration.

CASE 5.—A 9-week-old boy had respiratory difficulty since birth. A week preceding admission the infant had increasing air hunger manifest by rapid respiration and cyanosis on exertion. Examination gave evidence of a right tension pneumothorax. Roentgenographically there was gross mediastinal displacement to the left, with only a few lung markings on the right side (Fig. 5, *A*). A bronchogram showed compression of the entire right lung along the mediastinum (Fig. 5, *B*).

Operation was performed on Feb. 10, 1950, and a large multiloculated cyst of the right lower lobe was found. It was necessary to sacrifice portions of the posterior and lateral basal segments of the lobe due to the attenuation of lung parenchyma in those areas. The cyst wall was excised. During the closure of the bronchiolar orifices complete obstruction of the airway developed, the infant became cyanotic, and cardiac arrest occurred. A tracheotomy was started but before it was completed the airway obstruction was relieved, possibly by the hyperextension of the neck. Manual cardiac massage had been maintained and the heart promptly regained its normal rhythm. The chest was quickly closed about a catheter drain for suction and no further bronchiolar closure was accomplished, but it was felt the main orifices had been closed. Histologic study of the cyst wall showed the lining to be composed of respiratory epithelium and connective tissue. A persistent air leak occurred for nine days postoperatively. This sealed spontaneously and the infant was discharged on the twenty-first postoperative day with the lung expanded except for a small residual basal air-pocket. Twenty-three days following discharge he was re-admitted with findings of a right tension pneumothorax. Suction drainage of the pleural space was established, the lung promptly re-expanded. Drainage was discontinued after seventy-two hours, the infant was discharged fourteen days after admission with findings of a completely re-expanded lung.

Comment.—This patient illustrates the early age at which these lesions occur. The histologic finding of both respiratory epithelium and connective tissue in the lining of the cyst suggests that the rapid expansion of the cyst after birth may have ruptured the epithelial lining in the ballooning process.

The occurrence of a tension pneumothorax following discharge suggests either bronchial leakage from the site of the removal of the cyst or the rupture of an additional bleb. It was thought inadvisable again to explore the thorax in this infant in view of the prompt and satisfactory response to suction alone.

SUMMARY

Symptoms produced by single large cysts of the lung are extremely variable but depend upon compression of residual nondiseased lung tissue, superimposed infection, hemorrhage, and spontaneous rupture of the cyst. The response to treatment depends upon the presence or absence of a lining of respiratory epithelium. In the presence of an epithelial lining obliteration of the cyst cannot be anticipated until the bronchiolar fistulas are closed and the lining of the cyst removed. It is not possible to determine the presence or absence of an epithelial lining clinically. It is recommended that single large cysts of the lung be treated by simple excision of the walls of the space, closure

of bronchiolar fistulas, and obliteration of the pleural space by re-expansion of the lung with catheter drainage and suction. Cases to support these recommendations are cited.

ADDENDUM

Since this paper was submitted for publication, three additional cases of giant cyst of the lung have been operated upon using the method described. One patient developed a postoperative hemothorax which responded to aspiration. There were no complications in the other cases, and all three patients have satisfactory results.

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EXPERIMENTAL TUBERCULOSIS

II. Effects of Ligation of Pulmonary Arteries on Tuberculosis in Monkeys

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IN A previous report[1] we have described the effects of anastomosis between systemic and pulmonary arteries in monkeys with induced tuberculosis. As a further phase in a systematic investigation of experimental alteration of the circulation in pulmonary tuberculosis, this report is concerned with the effects of ligation of pulmonary arteries on tuberculosis in monkeys.

METHODS

In these experiments 23 healthy *Macaca mulatta* monkeys were used. All were selected from carefully segregated stock animals which had consistently shown negative reactions to repeated tuberculin tests. Into each animal was injected through the saphenous vein a standard suspension of the same virulent strain of *Mycobacterium tuberculosis hominis* which was used in our previous experiments.

The infecting dose was given to one group of monkeys 18 to 24 hours before operation, and a comparable group of animals were infected 6 to 13 days after operation. In both groups of animals the operation consisted of (a) division of right or left pulmonary artery, or (b) division of branches of the pulmonary artery to the right or left upper lobe, and (c) control thoracotomy.

BACTERIOLOGIC TECHNIQUE

A virulent strain of *Myco. tuberculosis hominis* isolated by one of us (B. J. O.) at the National Institutes of Health was used in these experiments. The virulence of this strain, the details of culture, and the standardized technique employed in preparation of emulsions for animal inoculation have been discussed in a previous communication.[1] The dose of tubercle bacilli given intravenously to each monkey in the present experiments consisted of 1 c.c. of a suspension containing 10^{-4} milligrams of organisms. As may be seen in Table I the number of injected viable particles (single or clumped bacilli) ranged from 99 to 387. This degree of variation occurred despite every effort to insure uniformity in different preparations of emulsions during the 15 month period in which these animal inoculations were made.

OPERATIVE TECHNIQUE

Intravenous sodium pentobarbital anesthesia (30 mg. per kilogram) with morphine-atropine premedication was used. Pulmonary ventilation was maintained by means of a mechanical respirator which delivered oxygen through an endotracheal tube.[2] Aseptic surgical technique was used in all operations. After the hair had been clipped from the operative area, the skin was painted with tincture of iodine followed by alcohol. The chest was entered anteriorly through the fourth intercostal space on the left.

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In ligation of the pulmonary artery the vessel to the exposed lung was dissected free in the mediastinum and divided between ligatures.* In ligation of branches of the pulmonary artery to the upper lobe the artery to the exposed lung was dissected free in the mediastinum, and the superior branch of the artery was isolated, doubly ligated, and divided. Dissection was carried distally along the descending trunk of the pulmonary artery and any additional branches entering the upper lobe were divided. A small recurrent branch entering the inferior aspect of the hilum of the right upper lobe was regularly encountered in these monkeys.

In control thoracotomy the exposed lung was packed down with moist gauze for 15 to 60 minutes. In addition, in some of these animals the pulmonary artery was dissected free and occluded by a rubber shod clamp for a brief interval.

After each type of operation the lung was carefully re-expanded and closure of the chest wall was carried out in layers with interrupted silk sutures. Following operation each monkey was given intramuscularly 300,000 units of penicillin in oil. The animals were kept in separate cages in the same isolation room and were fed stock diet until their deaths.

RESULTS

There were no immediate postoperative deaths in the 23 animals submitted to operation. All wounds healed per primam and there was no instance of wound infection or empyema. Tuberculosis developed in each of the 23 monkeys and the disease was allowed to progress to a fatal termination.† A complete autopsy was performed immediately after death in each case. A special attempt was made to compare the character and extent of the tuberculous process in the lobe or lung on the side of arterial interruption with the lesions in the other pulmonary units of the same animal and with the lesions in the lungs of the control animals. Sections were taken from the lungs and other viscera for microscopic study. Color photographs were made as an aid in recording gross pathologic appearances.

Results Following Ligation of Right or Left Pulmonary Artery.—In 8 monkeys the right or left pulmonary artery was ligated. In 4 of these animals the infecting dose of tubercle bacilli was given 18 to 24 hours before operation. In the other 4 monkeys the bacilli were injected 6 to 13 days after operation. The monkeys lived from 66 to 376 days after inoculation. Pertinent data for these animals are summarized in Table I.

In 7 of the 8 animals with ligation of the pulmonary artery the extent of the pulmonary disease as seen at autopsy was greater in the lung on the side of the arterial ligation. Examples of the differences in the disease process in the two lungs are depicted in Figs. 1, 2, 3, and 4. In general, there were numerous conglomerate tuberculous lesions in the lung on the side of operation. This lung showed massive caseous areas in contrast to the opposite lung which presented smaller, less widely distributed tubercles, usually of the miliary type. In 6 of the 8 animals the lung on the side of operation was larger than the opposite lung. This increase in size is largely attributable to the greater amount of caseous tuberculosis in the lung. Figs. 1 and 3 are examples of this change in pulmonary volume due to massive tuberculous infiltration.

*Throughout this report the term "ligation" will be used to indicate double ligation and division between the ligatures.

†One monkey died of an undetermined cause 18 days after ligation of the arteries to the right upper lobe. Autopsy showed only a few scattered miliary lesions in the liver.

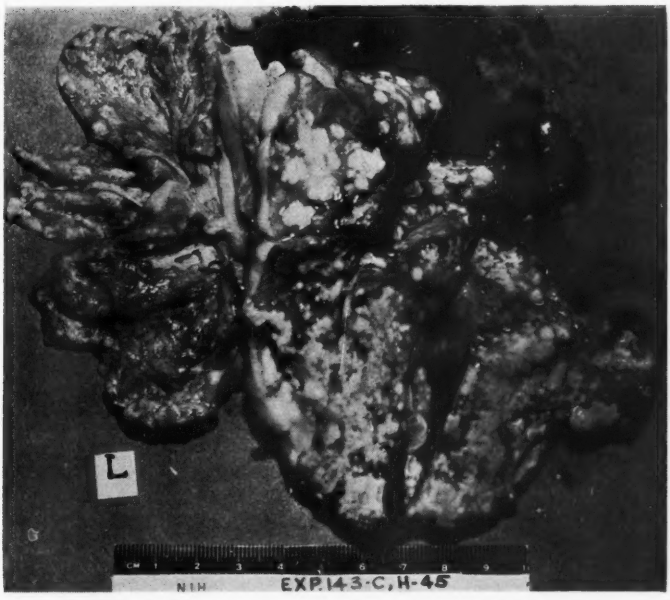

Fig. 1 (Monkey H-45).—Ligation of right pulmonary artery. Intravenous injection of tubercle bacilli on day preceding operation. Death 117 days later. Photograph of lungs at autopsy, showing confluent caseous tubercles in right lung; left lung shows only scattered miliary lesions.

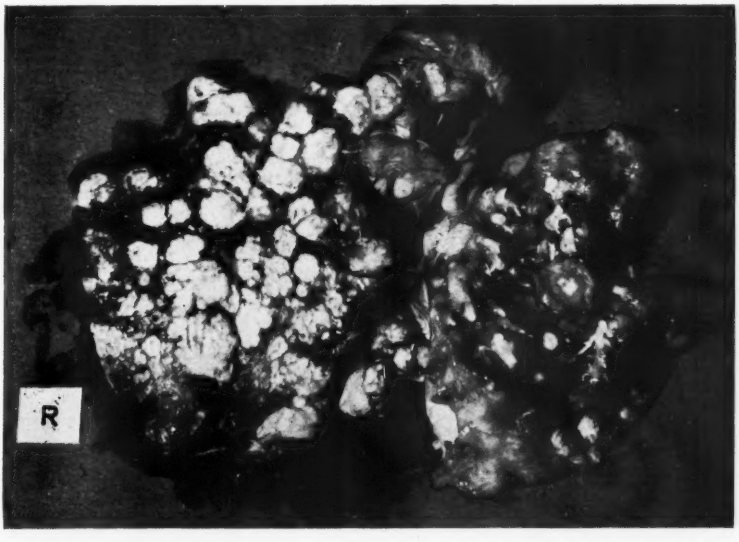

Fig. 2 (Monkey E-10).—Ligation of right pulmonary artery. Intravenous injection of tubercle bacilli on day preceding operation. Death 134 days later. Photograph of lungs at autopsy showing multiple large caseous foci in right lung. Tuberculous lesions are present in left lung but are smaller and less extensive than on the right. The projections from the cut surface of the left lung represent branches of the pulmonary artery filled with an injection mass introduced into the right ventricle at autopsy.

TABLE I. EXPERIMENTAL DATA ON EFFECTS OF LIGATION OF PULMONARY ARTERIES ON TUBERCULOSIS IN MONKEYS

MONKEY NUMBER	OPERATIVE PROCEDURE	DOSAGE OF TUBERCLE BACILLI (NUMBER VIABLE PARTICLES)	SURVIVAL TIME AFTER INJECTION (DAYS)	DISTRIBUTION OF PULMONARY LESIONS AT AUTOPSY		
				EXTENSIVE CASEATION IN LOBE OR LUNG WITH ARTERY LIGATED	MORE EXTENSIVE ELSEWHERE IN LUNGS	NO DIFFERENCE IN LOBES OR LUNGS
		Group Injected 18 to 24 Hours Before Operation				
E-10	Division right pulmonary artery	108	134	++++ Right lung		
H-45	Division right pulmonary artery	387	117	++++ Right lung		
H-21	Division left pulmonary artery		84	++ Left lung		
E-15	Division left pulmonary artery	164	376	++++ Left lung		
F-8	Division left upper lobe artery	176	162	++ Left upper lobe		
F-7	Division right upper lobe artery	164	177	+++ Right upper lobe		
H-46	Division right upper lobe artery	387	76	++ Right upper lobe		
H-44	Division right upper lobe artery	387	19			(No gross tuberculosis)
N-42	Control thoracotomy, left	108	98			Miliary
E-34	Control thoracotomy, right	108	76			Miliary
H-49	Control thoracotomy, right	387	131			Miliary
H-50	Control thoracotomy, right	387	119			Miliary
		Group Injected 6 to 13 Days After Operation				
H-33	Division left pulmonary artery	387	111	++++ Left lung		
H-34	Division left pulmonary artery	387	78	++++ Left lung		
H-35	Division left pulmonary artery	387	126	++++ Left lung		
H-17	Division left pulmonary artery		66			Miliary
H-9	Division right upper lobe artery	99	137	++++ Right upper lobe		
H-36	Division right upper lobe artery	387	84			Miliary
H-37	Division right upper lobe artery	387	72	+ Right upper lobe		
H-38	Division right upper lobe artery	387	90	+++ Right upper lobe		
H-6	Control thoracotomy, right	99	118			Miliary
H-42	Control thoracotomy, left	387	91			Miliary
H-43	Control thoracotomy, left	387	150			Miliary

In one monkey there was no apparent difference in the distribution or character of the lesions in the two lungs. This animal died 79 days after ligation of the left pulmonary artery and 66 days after the infecting dose of tubercle bacilli was given. At autopsy scattered small tubercles were observed in both lungs with an equal distribution of lesions.

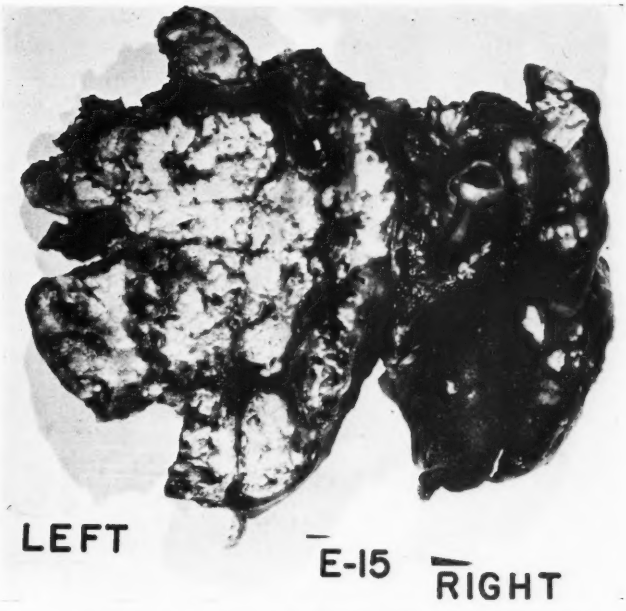

Fig. 3 (Monkey E-15).—Ligation of left pulmonary artery. Intravenous injection of tubercle bacilli on day preceding operation. Death 376 days later. Sectioned lungs photographed after fixation showing extensive caseation of lung on side of arterial ligation.

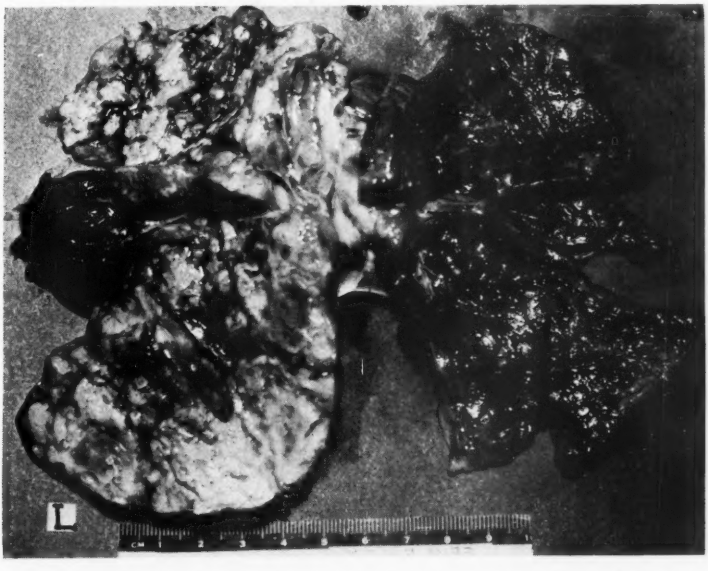

Fig. 4 (Monkey H-33).—Ligation of left pulmonary artery. Intravenous injection of tubercle bacilli 13 days after operation. Death 111 days later. Photograph of sectioned lungs at autopsy. The disease process is more extensive in the lung on the side of arterial ligation despite the fact that the initially injected tubercle bacilli were deposited primarily in the opposite lung by way of the pulmonary artery.

The extrapulmonary lesions in this group of monkeys were concentrated chiefly in liver, lymph nodes, and spleen, although scattered miliary lesions were distributed throughout many other viscera. Brain and meninges showed a few scattered miliary tubercles in 2 of these animals. Fig. 5 illustrates the lesions characteristically present in liver and spleen.

Microscopic study of sections from the lungs and other viscera of these monkeys confirmed the condition found at autopsy. Acid-fast stains demonstrated large numbers of tubercle bacilli in the lesions. There was a uniform lack of fibrosis around the areas. Microscopic examination of the lesions in the two lungs failed to show any significant differences which were not apparent from the gross examination.

Fig. 5.—Photograph of abdominal viscera in a monkey with division of pulmonary artery to right upper lobe. Death 84 days after infection. The same general distribution is seen in all animals of these groups, although the liver is often more heavily involved.

Results Following Ligation of Branches of Pulmonary Artery to Right or Left Upper Lobe.—The branches of the pulmonary artery to the upper lobe of the right or left lung were ligated in 8 monkeys. In 4 of these animals the infecting dose of tubercle bacilli was given 18 to 24 hours before operation. In the other 4 monkeys the bacilli were injected 6 to 13 days after operation. One of these latter animals died 18 days after operation of an undetermined cause; autopsy showed only a few scattered 1 mm. tubercles in the liver. The other 7 monkeys lived from 72 to 177 days after injection, and in each animal extensive tuberculosis was found at autopsy. Pertinent data for these animals are summarized in Table I.

In 6 of the 8 animals with ligation of branches of the pulmonary artery to the upper lobe the extent of the pulmonary disease as seen at autopsy was greater in the upper lobe on the side of the ligation. Examples of the difference between the disease process in the affected upper lobe and that in the remainder of the lungs are shown in Figs. 6, 7, 8, and 9.

Fig. 6 (Monkey H-38).—Ligation of pulmonary arteries to upper lobe, right lung. Intravenous injection of tubercle bacilli 13 days after operation. Died 90 days later. Disease process more extensive in right upper lobe.

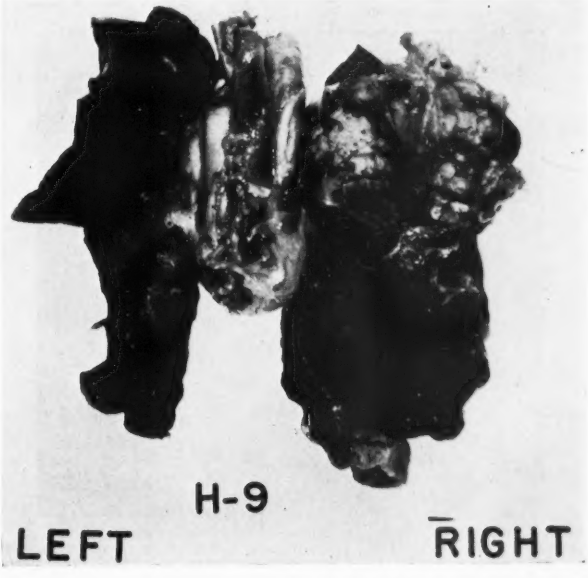

Fig. 7 (Monkey H-9).—Ligation of pulmonary arteries to upper lobe, right lung. Intravenous injection of tubercle bacilli 6 days after operation. Died 137 days later. Disease process more extensive in right upper lobe.

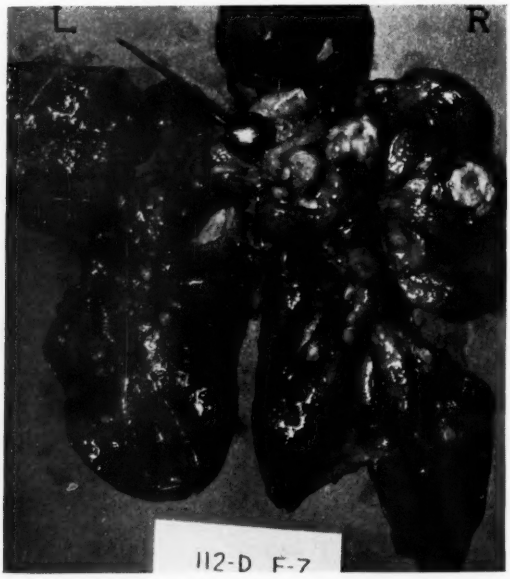

Fig. 8 (Monkey F-7).—Ligation of pulmonary arteries to upper lobe, right lung. Intravenous injection of tubercle bacilli 18 hours before operation. Died 177 days later. Large caseous foci in right upper lobe.

Fig. 9 (Monkey F-8).—Ligation of pulmonary arteries to upper lobe, left lung. Intravenous injection of tubercle bacilli 20 hours before operation. Died 162 days later. Large caseous focus in left upper lobe.

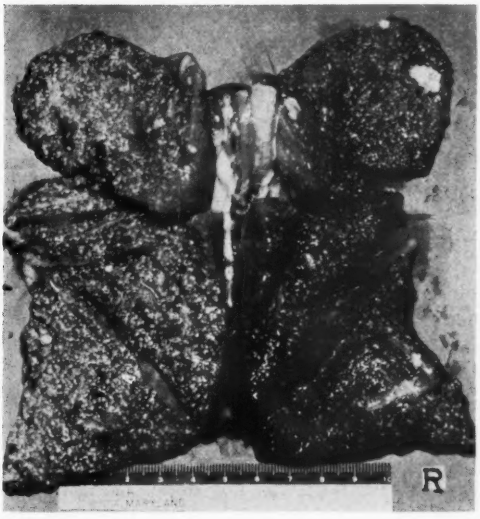

Fig. 10*A* (Monkey H-50).—Control thoracotomy, right. Intravenous injection of tubercle bacilli on day before operation. Died 119 days later. No essential difference between the lungs.

Fig. 10*B* (Monkey N-42).—Control thoracotomy, left. Intravenous injection of tubercle bacilli on day before operation. Died 98 days later. Lungs show equally distributed miliary lesions.

In general, the same changes in the disease process were found in the affected upper lobes of these animals as were encountered in the entire lung of the monkeys with ligation of the pulmonary artery. There were large conglomerate caseous masses in the upper lobe with arterial branches ligated in contrast to scattered small lesions in the remainder of the lung.

In 2 animals the disease process was of no greater extent in the upper lobe with ligated arteries. One of these monkeys received the infecting dose of organisms 13 days after the arteries to the upper lobe had been ligated. At autopsy there were seen symmetrically distributed small lesions throughout all lobes of both lungs. The second animal died so soon after inoculation (19 days) that there was no gross evidence of tuberculosis in the lungs at autopsy.

In these monkeys with ligation of arteries to the upper lobe the extrapulmonary lesions followed the pattern previously described in monkeys with ligation of the pulmonary artery. Microscopic study confirmed the observations made on gross examination. Tubercle bacilli were demonstrated in the lesions by acid-fast stains.

Results Following Control Thoracotomy.—In 7 monkeys thoracotomy was carried out but arterial ligation was not done. In all of these animals the lung was packed down for a brief period to simulate conditions existing during ligation of a pulmonary artery. Moreover, in 2 monkeys the pulmonary artery was dissected free and was occluded by a rubber shod clamp for a short time. Four of these monkeys were given the infecting dose of tubercle bacilli 18 to 24 hours before operation; the other 3 monkeys were infected 6 to 13 days after operation. These control animals lived from 76 to 150 days after inoculation. Results are summarized in Table I.

At autopsy the lungs of these 7 monkeys showed an equal distribution of tubercles of miliary type, ranging from 1 to 5 mm. in diameter. There were no observable differences between the two lungs from the standpoint of character or extent of the disease process, nor was there any difference in the extent or distribution of pulmonary tubercles in the monkeys inoculated before operation and in those inoculated after operation. Typical findings at autopsy in the lungs of these animals are illustrated in Fig. 10.

Extrapulmonary lesions in these control animals were similar in character and distribution to those in the monkeys with arterial ligations. Microscopic study of the lesions in the control animals demonstrated numerous tubercle bacilli and confirmed the condition observed at autopsy.

DISCUSSION

The results of this investigation indicate that in monkeys with induced tuberculosis, ligation of a pulmonary artery produces in the area supplied by the artery a pathologic process which differs from that in the remainder of the pulmonary tissue. When the artery to one lung is ligated, the disease process becomes more extensive in the lung on the side of ligation. When a lobar artery is ligated, the disease process in that lobe becomes more extensive than in the remaining lobes. Conglomerate caseous tuberculous lesions are found in

the pulmonary unit thus deprived of a portion of its blood supply. In contrast, the remaining pulmonary tissue shows the miliary form of tuberculosis typically found in both lungs of control animals.

All of these animals were given the same dose (10^{-4} mg.) of the same strain of *Myco. tuberculosis hominis.* In all experiments the organisms were introduced through the saphenous vein. In half of the animals the infecting dose was given 18 to 24 hours before operation. This preoperative injection was designed to effect equal seeding of the two lungs with the initial dose of organisms.

In the remaining animals the organisms were injected 6 to 13 days after operation. It seems apparent that in those animals with ligation of the pulmonary artery to a lobe or to an entire lung, organisms injected after operation would be deposited in small numbers in the pulmonary unit thus deprived of its pulmonary arterial blood. Despite this smaller initial inoculum the disease process at autopsy was in most instances found to be more extensive in the lobe or lung with a ligated pulmonary artery. A summary of the over-all results of ligation of pulmonary arteries in both groups of monkeys is presented in Table II.

TABLE II. RESULTS OF LIGATION OF PULMONARY ARTERIES IN SIXTEEN MONKEYS WITH INDUCED TUBERCULOSIS

Lesions more extensive in lobe or lung with artery ligated	13
No difference in distribution of pulmonary lesions	2
Early death without gross pulmonary lesions	1

These effects of pulmonary artery ligation may be considered in relation to previous observations on this subject. In 1924, Schlaepfer[3] studied the changes in the uninfected lung of the dog after ligation of a pulmonary artery. On the basis of the fibrosis and shrinkage which occurred he concluded that ligation of a pulmonary artery might prove to be an effective means of treatment in human pulmonary tuberculosis. Hesse[4] has recently reported that partial occlusion of the main pulmonary arterial trunk resulted in increased susceptibility to tuberculosis in guinea pigs exposed to air-borne infection.

A few attempts have been made to modify the course of human pulmonary tuberculosis by ligation of a pulmonary artery. In 2 patients with cavitary tuberculosis Wilms[5] ligated the pulmonary artery with apparently beneficial results. Eloesser[6] in a report on surgical attempts at relief of severe hemoptysis in pulmonary tuberculosis, described a patient who was relieved of hemoptysis for 8 months after ligation of the pulmonary artery to the right upper lobe. Hemorrhage then recurred and the ultimate fate of the patient was not recorded. Rienhoff[7] in a recent publication mentioned a group of 8 patients with pulmonary tuberculosis in whom ligation of a pulmonary artery resulted in disastrous extension of the disease.

The results of the present experiments in monkeys with induced tuberculosis, together with the unfavorable results reported by Rienhoff, suggest strongly that ligation of a pulmonary artery is inadvisable in the surgical treatment of human pulmonary tuberculosis.

The changes produced in the disease process in this group of monkeys by ligation of pulmonary arteries are essentially the same as those which were encountered during an earlier phase of this investigation in the lungs of monkeys with diversion of systemic blood into a pulmonary artery. In the previous study[1] it was found that anastomosis between systemic and pulmonary arteries in monkeys with intravenously induced tuberculosis resulted in an extensive caseous form of the disease in the lung on the side of the anastomosis.

It is interesting that the same extensive caseous changes develop in the lung of monkeys with induced tuberculosis which undergo such widely different operative procedures as ligation of a pulmonary artery and anastomosis between a systemic and a pulmonary artery. These procedures appear to produce in the lung on the side of operation similar favorable conditions for progression of the disease.

A lung with a ligated pulmonary artery is supplied only by the bronchial artery. In contrast, a lung with a systemic-pulmonary anastomosis is supplied not only by the bronchial artery but by the arterial blood flowing through the anastomosis. There is obviously a greater blood flow to the lung under the latter condition. In both cases, however, the lung on the side of operation is perfused only by fully oxygenated blood in contrast to a normal lung which receives a mixture of oxygenated and unoxygenated blood. Thus, it is possible that the caseous pulmonary changes encountered in monkeys with pulmonary artery ligation and in those with systemic-pulmonary anastomosis are related to high oxygen tension in the blood perfusing the lung on the side of operation. Further work is required to evaluate the importance of this and other factors which may be related to these changes in the tuberculous process.

SUMMARY

In a series of 23 monkeys tuberculosis was induced by intravenous injection of human tubercle bacilli. The effect of ligation of the pulmonary arteries in these animals has been observed. In 7 of 8 monkeys with interruption of the pulmonary arterial blood supply to an entire lung there resulted an extensive, caseous tuberculous process in that lung, in contrast to the scattered miliary lesions found in the opposite lung.

Similarly, when the pulmonary artery to a single lobe was ligated, an extensive caseous process developed in that lobe in 6 of 8 animals. In contrast, scattered miliary lesions were found in all other lobes on both sides.

In 7 monkeys with induced tuberculosis which were submitted to simple thoracotomy, the tuberculous process was uniformly miliary in type with no difference between the lung on the side of operation and the opposite lung.

There is no essential difference between a group of monkeys given the infecting dose of organisms on the day preceding operation and a comparable

group infected 6 to 13 days after operation. The caseous tuberculous process which was observed after ligation of pulmonary arteries is similar to that which we have described after anastomosis between systemic and pulmonary arteries in monkeys. The possible relation of these changes to increased oxygenation of the blood perfusing the lung in these two instances of altered pulmonary circulation is noted.

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PULMONARY FUNCTION BEFORE AND AFTER EXTRAPLEURAL PNEUMOTHORAX

A Comparison With Other Forms of Collapse and Resection

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THE primary aim of any collapse procedure or resection in the treatment of pulmonary tuberculosis is arrest of the disease. Operative mortality and morbidity, complications or deformities, and late statistics further influence the fate of any particular method of treatment. In the choice of procedure preservation of pulmonary function as another factor has been increasingly recognized during the past twenty years. Collapse and resection procedures which are most selective and interfere least with ventilation are now known to carry a smaller operative mortality and are followed by a smoother convalescence. Furthermore, such "parenchymal-sparing" operations can be made available to a group of patients with very advanced or bilateral disease whose pulmonary reserve is such that only the most "selective" of procedures can be tolerated. Finally, such procedures cause less disability and therefore offer a greater opportunity for the patient to be restored to economic usefulness.

The effect on pulmonary function of many presently employed collapse procedures has been investigated. These reports to some extent have been responsible for the decline in popularity of such therapeutic measures as pneumothorax, and have prompted phthisiologists and thoracic surgeons to look for modifications of thoracoplasty in order to avoid paradoxical movement of the chest wall. Other forms of therapy, as pneumoperitoneum, have been received with great favor partly because of their complete reversibility and the small loss of pulmonary function incident to their administration.

The enthusiastic introduction of the modern extrapleural pneumonolysis followed by extrapleural pneumothorax by Schmidt[1] and Graf[2] was based to a great extent on the clinical observations of the "selectivity" of the procedure and the laboratory findings of Gaubatz[3] who showed that extrapleural pneumothorax caused relatively little loss of function.

Most investigators, since then, no matter what their opinion of the value of the operation as a whole, have agreed that extrapleural pneumothorax is selective and hence can be made available to patients whose pulmonary reserve is such that they could not tolerate thoracoplasty. They have further gen-

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erally observed that dyspnea after this operation is uncommon and a few who measured vital capacity before and after operation found the loss small. Similar observations concerning extrapleural plombage were made by Alexander sixteen years ago. He performed such an operation on a patient with a vital capacity of 800 c.c. without ill effect.[4] Like experiences have been reported recently when some of the newer plombage materials were used.[5]

The only recorded study of the effect of extrapleural pneumothorax on pulmonary function was made by Gaubatz in 1938.[3] Maximum breathing capacity, vital capacity, residual air, and oxygen debt were studied before and less than one month after operation in 77 patients. Improved function was reported after operation in 24 per cent of the patients, unchanged function in 33 per cent, and the remainder showed a loss of function of no more than 10 per cent. He compared these results with those after thoracoplasty and phrenic interruption and concluded that extrapleural pneumothorax and oleothorax caused less loss of function than either of the other operations.[6] No reports on the physiologic effect of extrapleural pneumothorax have appeared in this country.

We have undertaken to study the effects of extrapleural pneumothorax on pulmonary physiology for several reasons: (1) the advent of newer methods of study of residual air and of split function by bronchospirometry; (2) the availability for comparative purposes of a large number of other types of collapse procedures studied at the same postoperative intervals and by the same methods of investigation; (3) the possibility of comparing extrapleural pneumothorax not only with thoracoplasty but with medical collapse procedures and with newer types of treatment, particularly resection; (4) the paramount importance of comparing the effect of various collapse measures on lungs with comparable pathologic lesions; and (5) the recent renewed interest in extrapleural pneumothorax.[7, 8] This renewed interest has been stimulated by the availability of antibacterial agents,[9] the potential usefulness of the procedure as a preliminary to thoracoplasty,[10] and the increasing demand for more "selective" collapse procedures that will avoid the paradoxical movement of the decostalized chest wall and offer maximal preservation of function.

METHOD

Selection of Patients.—Extrapleural pneumothorax was carried out on 30 occasions on 28 patients with the following three indications (Table I): (1) previous contralateral collapse therapy with resulting contralateral limitation of function making the use of other collapse procedures inadvisable (12 cases). In general, we have hesitated to perform thoracoplasty or resection if the involved lung carried more than 60 per cent of the total function, no matter what the total function might be. (2) Bilateral cavitary disease of such nature that the necessity of eventual bilateral collapse therapy appeared great (13 cases). (3) As a preliminary to a modified type of thoracoplasty (5 cases) requiring extrapleural pneumothorax as a first stage, as used by Harken and Cincotti.[10]

Operative Procedure.—A standard extrapleural pneumonolysis through the bed of the fourth posterior rib was performed on all patients.[11, 12, 13] The dissection on the mediastinal aspect was carried down to and over the azygos vein on the right and the arch of the aorta

TABLE I. INDICATIONS FOR EXTRAPLEURAL PNEUMOTHORAX; VITAL CAPACITY AND RESIDUAL AIR BEFORE AND AFTER OPERATION

CASE NO.	AGE (YEAR)	SEX	CONTRALATERAL LUNG	VITAL CAPACITY (LITERS)				RESIDUAL AIR IN C.C.	
				NORMAL	PREOP.	14 DAYS POSTOP.	6 MONTHS POSTOP.	PREOP.	POSTOP.
			Group 1						
1	21	F	50% pneumothorax	3.25	1.92	1.56	1.60	—	—
2	31	F	70% hydropneumothorax	2.99	2.14	1.86	1.90	—	—
3	17	F	Pneumothorax, cavitary disease	3.37	1.81	1.58	—	—	—
4	36	F	Unexpandable pneumothorax lung	2.94	1.66	1.55	1.38	—	—
5	34	F	Pneumothorax, cavitary disease	2.91	2.01	2.00	2.00	—	—
6	34	F	Pneumothorax, cavitary disease	3.04	1.51	1.36	1.95	—	—
7	24	F	No lung	3.25	1.36	1.01	1.09	720	200
8	39	M	90% pyopneumothorax	3.88	2.78	2.22	2.48	1,100	480
9	44	M	10 rib thoracoplasty	4.25	1.88	1.56	—	1,650	920
10	23	M	Cavitary disease	4.36	3.20	2.55	2.85	1,240	820
11	23	M	Extrapleural pneumothorax	4.36	2.85	2.42	2.45	820	415
12	41	M	Effusion, atelectasis	3.97	2.90	2.10	2.39	—	—
13	35	M	Effusion, unexpanded lung	4.25	3.40	2.86	2.68	1,110	710
14	49	M	Cavitary disease	3.66	2.95	2.45	2.58	1,360	810
15	49	M	Extrapleural pneumothorax	3.66	2.58	2.01	—	810	625
16	39	M	Cavitary disease	4.09	3.13	2.45	2.43	—	—
17	33	F	Cavitary disease	3.00	3.17	2.34	2.60	—	—
18	40	M	Cavitary disease	3.95	3.73	3.16	3.20	1,400	910
19	50	M	10 rib thoracoplasty	3.92	1.70	1.52	—	—	—
20	37	M	Cavitary disease	4.23	2.70	2.35	—	—	—
21	22	F	Cavitary disease	3.32	2.32	1.99	—	—	—
22	61	M	"Normal"	3.85	3.05	2.95	3.06	—	—
23	40	F	"Normal"	2.88	3.03	2.58	2.5	1,620	1,105
24	37	F	"Normal"	2.80	2.15	1.84	1.90	970	715
25	46	M	"Normal"	3.90	3.65	3.12	3.26	1,100	820
26	20	F	"Normal"	3.39	2.98	2.43	2.80	1,005	510
27	26	M	Cavitary disease	4.16	1.68	1.58	—	—	—
Mean				3.59	2.77	2.25	2.42	1,145	695
			Group 2						
28	38	M	Surgical decortication	4.17	2.38	0.90	—	—	—
29	42	F	4 rib thoracoplasty	2.72	1.85	0.80	—	—	—
30	52	F	Cavitary disease	2.65	1.58	0.75	—	—	—
Mean				3.18	1.94	0.82	—	—	—

on the left. Postoperative care was identical to that by others except that all spaces were tapped dry assiduously and 500,000 units of penicillin and 0.5 Gm. of streptomycin were injected into the extrapleural space daily for two weeks.

Operative Results.—Four patients have had previously planned conversions to thoracoplasty. The space was lost in 4 patients for various reasons making postoperative management of the spaces unsatisfactory. The remaining 20 patients carry 22 extrapleural spaces. There have been no deaths, operative or otherwise, no bronchopleural fistulas, no extrapleural empyemas, and no spreads or reactivations.

Ventilatory Studies.—A detailed outline of ventilatory studies as performed in our laboratory has been reported elsewhere.[14] All patients were studied immediately prior to and fourteen days after operation and six months later after having been on walking exercises for two months. A few of the recently operated patients and those in whom thoracoplasty conversion was planned, were restudied as early as two months after operation.

Resting minute ventilation, vital capacity and its subdivisions, minute oxygen consumption, and the ventilatory equivalent were obtained from spirograms made with a closed circuit water spirometer with the patient breathing oxygen in the supine position. Vital capacities were also determined in the upright position. The maximum breathing capacity[15] was collected with a high velocity respiratory valve and a Douglas bag.[16] The walking ventilation was obtained in like manner during moderate exercise—walking on a level at the rate of 180 feet per minute for three minutes. The walking index was calculated according to the formula $\frac{\text{walking ventilation in liters/minute}}{\text{maximum breathing capacity in liters/minute}} \times 100$.[16] All tests were performed in triplicate and the best effort was recorded. Volumes were corrected to 37° C. Predicted normal values for vital capacity and maximum breathing capacity were calculated according to the regression formulas of Cournand and associates.[17] The air velocity index, previously described by one of us (E. A. G.), was calculated according to the formula $\frac{\text{per cent of predicted maximum breathing capacity}}{\text{per cent of predicted vital capacity}}$.[18] The normal value for this index is 1.0. Significantly smaller values indicate slowing of the expiratory air stream due to loss of elasticity of the lung or bronchial obstruction,[19] while larger values than 1.0 indicate greater than normal ventilatory efficiency. The latter are usually encountered with pathological or surgical loss of pulmonary parenchymal tissue. Residual air was determined with some modifications by the open circuit oxygen breathing method of Darling and associates.[20]

Bronchospirometry.—This was carried out immediately prior to operation in all but three patients. One of these had a previous contralateral pneumonectomy and the other two had severe recurrent hemoptyses continuing at the time of operation. The technique described by Jacobaeus[21] was carried out with a Zavod double-lumen catheter[22] and oxygen was supplied with a Gebauer twin spirometer.[23] Occasionally patients were allowed to breathe ambient air and the expired air was collected in Douglas bags and analyzed.[24] The same method was used in each patient before and after operation.

RESULTS

Results will be discussed in two sections. In the first, changes in pulmonary function will be reported after 27 operations during which the pleura was not opened, or, if opened, no intrapleural pneumothorax resulted either because of adhesions in that area or because of immediate postoperative intrapleural catheter drainage with underwater seal. The second group will deal with three cases in which the pleura was opened at operation with a resulting intrapleural hemopneumothorax which persisted for at least two weeks in spite of assiduous postoperative aspirations (Table I).

Group 1

Ventilatory Studies.—The mean maximum breathing capacity before operation was 84 liters per minute or 78 per cent of the mean predicted value. Fourteen days after operation it was 80 liters per minute and six months after operation 85 liters per minute. Thus postoperatively there was a drop of maximum breathing capacity of only 5 per cent and eventually there was no loss but a net gain. The great variability of functional results seen after thoracoplasty was here absent.[25] The largest single loss encountered at any time after operation was 17 per cent and that occurred in a patient who had had a previous contralateral pneumonectomy (Case 7, Table II).

The mean vital capacity before operation was 2.77 liters, or 77 per cent of the mean predicted figure. Preoperative ventilatory impairment, as meas-

TABLE II. MAXIMUM BREATHING CAPACITY, WALKING VENTILATION, AND WALKING INDEX BEFORE AND AFTER EXTRAPLEURAL PNEUMOTHORAX

CASE NO.	MAXIMUM BREATHING CAPACITY (L/MIN.)				WALKING VENTILATION (L/MIN.)	WALKING INDEX (PER CENT)		
	NORMAL	PREOP.	14 DAYS POSTOP.	6 MONTHS POSTOP.		PREOP.	14 DAYS POSTOP.	6 MONTHS POSTOP.
				Group 1				
1	96	71	72	74	20	28	28	27
2	84	85	87	88	18	21	21	20
3	106	57	60	—	22	39	37	—
4	85	72	71	73	17	24	24	23
5	86	65	65	68	13	20	19	19
6	90	58	53	73	17	34	—	23
7	100	65	54	56	21	32	39	32
8	109	83	63	78	17	20	27	22
9	143	52	48	—	21	40	44	—
10	123	115	96	119	16	14	17	13
11	123	119	108	107	16	15	15	15
12	116	61	57	67	18	30	—	27
13	122	102	94	91	13	13	16	17
14	98	90	90	91	14	16	16	16
15	98	91	86	—	14	16	18	—
16	134	131	121	126	16	12	13	12
17	101	90	88	101	16	18	18	16
18	112	121	108	116	17	14	18	17
19	109	39	37	—	16	41	43	—
20	142	66	62	—	28	42	45	—
21	91	64	61	—	18	28	29	—
22	121	121	119	121	23	19	19	19
23	98	85	78	88	19	22	24	21
24	82	77	86	84	14	18	13	14
25	123	102	103	113	24	23	23	21
26	102	105	104	125	15	14	14	12
27	123	51	53	—	12	24	23	—
Mean	108	84	80	85	18	24	25	23
				Group 2				
28	107	56	20	—	18	32	90	—
29	69	56	36	—	18	32	50	—
30	74	70	43	—	18	26	42	—
Mean	83	61	33	—	18	30	61	—

ured by vital capacity and maximum breathing capacity, was roughly the same. After operation the mean vital capacity dropped to 2.25 liters, a loss of 18 per cent, and six months after operation it was 2.42 (Table I).

The residual air, measured in 13 cases, showed a rather large decrease from a mean of 1.15 liters before operation to 0.69 liters after operation, a loss of 40 per cent. It is apparent that the extrapleural space is created chiefly at the expense of residual air (Table I).

The mean air velocity index (Table III) was slightly elevated to 1.12 before treatment, a not unexpected finding since many of the patients carried partially collapsed lungs preoperatively and all had advanced parenchymal disease.[18] Seven patients showed indices of less than 1.0. Bronchial asthma was ruled out in these by bronchodilator studies.[19] Pulmonary emphysema was confirmed by residual air determinations in three of the patients. Since there was no appreciable loss of maximum breathing capacity after operation, the air velocity index rose to 1.27 and showed a further small increase during the next few months. In other words, although the patients' lung volumes

TABLE III. BRONCHOSPIROMETRY AND AIR VELOCITY INDEX BEFORE AND AFTER EXTRAPLEURAL PNEUMOTHORAX

CASE NO.	BRONCHOSPIROMETRY, PER CENT FUNCTION OF OPERATED LUNG						AIR VELOCITY INDEX		
	OXYGEN UPTAKE		VENTILATION		VITAL CAPACITY			14 DAYS	6 MONTHS
	PREOP.	POSTOP.	PREOP.	POSTOP.	PREOP.	POSTOP.	PREOP.	POSTOP.	POSTOP.
				Group 1					
1	77	71	75	62	72	62	1.25	1.56	1.57
2	72	65	58	59	74	67	1.42	1.66	1.64
3	34	34	38	39	36	36	1.00	1.21	—
4	65	55	64	59	73	58	1.52	1.65	1.83
5	45	43	55	53	56	54	1.10	1.15	1.15
6	—	—	—	—	—	—	1.28	1.31	1.27
7	100	100	100	100	100	100	1.55	1.74	1.65
8	85	82	79	73	89	84	1.07	1.02	1.12
9	86	86	65	58	72	69	0.82	0.92	—
10	77	53	74	55	72	52	1.29	1.34	1.49
11	47	36	45	35	48	39	1.49	1.57	1.58
12	49	33	58	35	52	31	0.73	0.92	0.97
13	53	46	61	46	65	44	1.05	1.15	1.19
14	52	50	49	40	54	41	1.14	1.37	1.31
15	50	51	60	51	59	51	1.31	1.60	—
16	50	45	22	18	30	26	1.29	1.50	1.58
17	40	35	51	44	46	35	0.84	1.11	1.15
18	34	34	37	18	36	23	1.14	1.20	1.29
19	96	91	68	65	80	79	0.84	0.87	—
20	—	—	—	—	—	—	0.76	0.77	—
21	36	32	34	30	29	24	1.00	1.12	—
22	35	29	44	43	45	41	1.27	1.29	1.27
23	38	37	45	40	39	39	0.83	0.89	1.00
24	36	36	57	51	38	42	1.22	1.59	1.50
25	38	37	56	47	46	37	0.88	1.05	1.09
26	40	34	52	38	46	36	1.17	1.42	1.48
27	—	—	—	—	—	—	1.05	1.27	—
Mean	56	51	56	48	57	49	1.12	1.27	1.29
				Group 2					
28	72	—	62	—	80	—	0.91	0.86	—
29	70	—	66	—	72	—	1.19	1.79	—
30	11	—	24	—	15	—	1.58	2.07	—
Mean	51	—	51	—	56	—	1.23	1.57	—

had decreased, they showed no loss of maximal ventilation and were therefore able to exchange more rapidly and more efficiently.[18]

The mean walking ventilation of 18 liters per minute corresponded fairly closely to the patients' metabolic requirements as expressed by surface area.[14] Only one patient (Case 20) showed an abnormally high ventilation during exercise. There was no significant change of walking ventilation after operation. The mean walking index, or percentage of maximum breathing capacity, which patients were required to use during this exercise was 24 per cent before operation. Several patients had indices of over 40 per cent and were therefore close to dyspnea at rest.[16] The mean walking index six months after operation was 23 per cent. In none of these patients, therefore, did extrapleural pneumothorax result in a disadvantage from the ventilatory standpoint.

Bronchospirometry.—Preoperative split function studies remarkably showed a larger share of the total function being carried by the lung to be operated upon. This was due to the fact that 12 of the 27 patients had had contra-

lateral collapse therapy at the time of study, and most others had contralateral cavitary disease or effusion with unexpanded lungs. Only 4 of the group of 26 had "normal" contralateral lungs of the type one prefers to see prior to resection or thoracoplasty. The mean oxygen uptake, as per cent of the total, of the lungs to be collapsed was 56 per cent, the mean minute ventilation also was 56 per cent, and the mean vital capacity was 57 per cent of the total. These same values after operation were 51, 48, and 49 per cent, respectively. There was, therefore, a shift of function to the contralateral side of 5 per cent for oxygen uptake and of 8 per cent for ventilation and vital capacity. Even after operation, therefore, the operated lungs carried half of the total function.

Comparison With Other Collapse Procedures and Resection.—We have chosen to compare the preceding results with those obtained in our own laboratory after other operations rather than with those reported in the literature. This was done because we felt that although the testing procedures used by us are well standardized, nevertheless, variations in detail of technique from one laboratory to another are inevitable. Furthermore, all of our patients were studied at the same times—immediately preoperatively, 14 days and 6 months after operation. This made for more valid comparison. Last, many reports in the literature dealt with loss of function of individual patients and mean values but usually disregarded the type of lung on which the operation was performed. This, in our experience, is the single most important factor, as far as postoperative functional results are concerned—far more important than the number of ribs taken at thoracoplasty, or even the type of procedure itself.[25]

In Fig. 1 the functional losses 14 days after operation are compared regardless of the type of lung on which the operation was performed. The most severe impairment of ventilatory function was due to thoracoplasty in 121 instances with a loss of 26 per cent for maximum breathing and 35 per cent for vital capacity. This large loss immediately after completion of stages is partly due to collapse, and partly to paradoxical chest motion which is usually marked so soon after operation. Loss of function after 24 pneumonectomies and 20 lobectomies was almost as large as that after thoracoplasty. Phrenic interruption resulted in a 10 per cent decrease of maximum breathing capacity and an 18 per cent loss of vital capacity. Extrapleural pneumothorax, with a maximal space at this early postoperative period caused the least loss of ventilatory function—5 per cent for maximum breathing capacity and 18 per cent for vital capacity.

Of greater importance than the fourteen-day studies are the functional end results, as measured six months after operation (Fig. 2). Here the functional loss after pneumothorax was by far the greatest, larger than the loss with thoracoplasty 14 days postoperatively. Pneumothorax was not studied fourteen days after induction, since the desired shape and extent of collapse usually had not been obtained at that time. The functional end result of thoracoplasty was much better than the immediate result. Losses for both vital capacity and maximum breathing capacity were only one-half as large

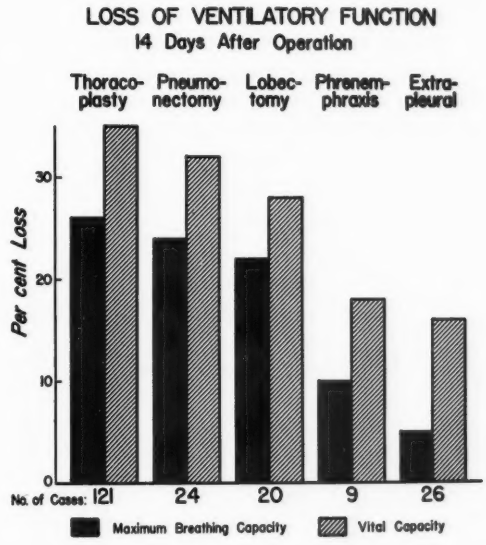

Fig. 1.—Per cent loss of maximum breathing capacity and vital capacity for various collapse procedures and resection 14 days after operation.

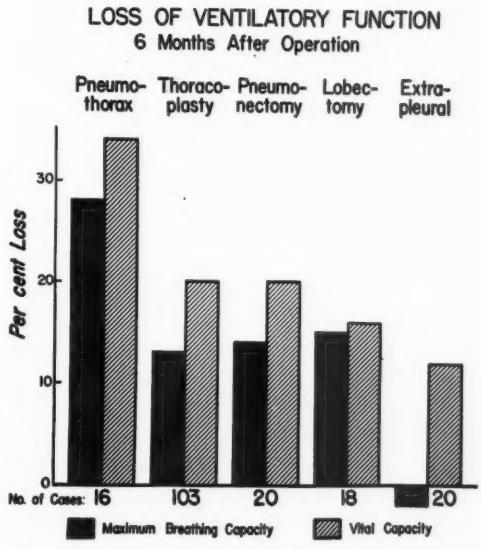

Fig. 2.—Per cent loss of maximum breathing capacity and vital capacity for various collapse procedures and resection 6 months after operation.

as immediately after operation. Patients with lobectomy or pneumonectomy showed considerable improvement during the further postoperative period, but this was not so marked as with thoracoplasty. Patients after phrenic interruption were studied six months after operation but motion of the diaphragm had usually returned by that time and results were therefore not included. Extrapleural pneumothorax patients, six months after operation, show a smaller decrease of vital capacity than after any of the other procedures and extrapleural pneumothorax was the only operation which caused no loss, but a small gain, of maximum breathing capacity.

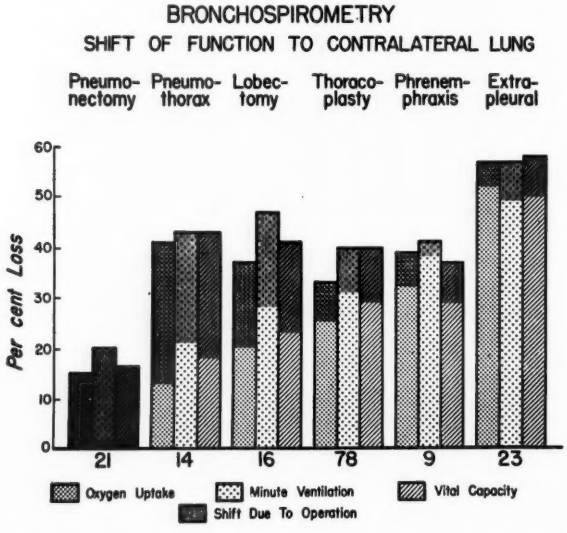

Fig. 3.—Shift of function to contralateral lung due to various collapse procedures and resection as determined by preoperative and postoperative bronchospirometry. The total height of the blocks represent share of total oxygen uptake, ventilation, and vital capacity of lungs before operation and the shaded areas represent shift of function to contralateral side due to operation.

The preceding studies are of interest in that they show what type of results were obtained with the various procedures as they were applied to individual cases. They are of no value for actual comparison since the type of lung for which the operations were performed were disregarded. The more nearly normal the function of the lungs, the larger the loss of function after collapse, no matter how collapse is obtained. This obvious fact has been shown by others and in our own laboratory.[25] Furthermore, the choice of procedure in many cases depended upon the share of function carried by the diseased lung. This is illustrated in Fig. 3. The total length of the blocks represents the share of the function-oxygen uptake, ventilation, and vital capacity—carried by the lung to be collapsed or resected. The shaded areas represent the shift of function to the other lung due to operation, as determined by broncho-

spirometry. It is apparent that most of our patients selected for pneumonectomy had almost useless lungs from a functional standpoint, and pneumonectomy was elected as the procedure of choice in many cases for that reason. The average share of the total oxygen uptake of pneumonectomy lungs was only 15 per cent. The function of lungs chosen for pneumothorax, lobectomy, and thoracoplasty varied from 30 to 40 per cent. At the other extreme were the extrapleural lungs with an average preoperative function of over 50 per cent. The shaded areas representing loss of relative function obviously cover the entire pneumonectomy area. Losses after pneumothorax, lobectomy, thoracoplasty, and phrenic interruption were strictly comparable since the operations were performed on the same type of lungs. These functional losses were greatest by far for pneumothorax and smallest for phrenic interruption.

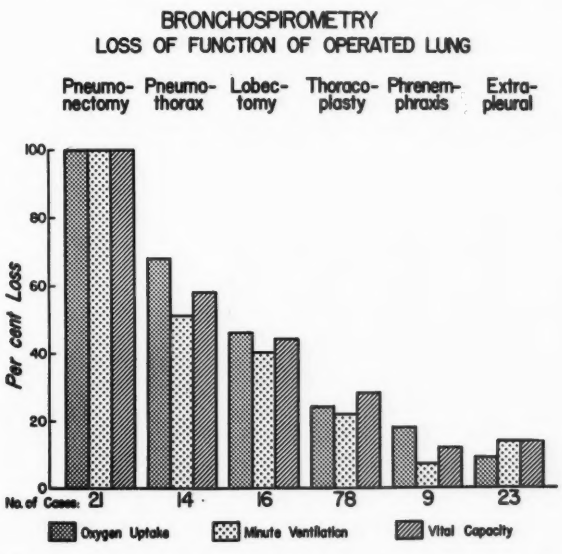

Fig. 4.—Function of lung to be operated upon was considered 100 per cent, no matter what the actual share of the function. The height of the blocks represent per cent loss of oxygen consumption, ventilation, and vital capacity after operation.

This last operation resulted in no appreciable loss of resting ventilation. This was also demonstrated by one of us (E. A. G.) with the aid of bronchospirometry during electrophrenic respiration.[26] The shift of function after extrapleural pneumothorax was relatively the smallest of all and the total shift compared favorably with that after phrenic interruption.

Comparison can be made in a different and perhaps more pertinent way from the same data: the function of the lung to be operated upon can be considered 100 per cent, no matter what the actual magnitude. From postoperative studies the per cent of loss of function of the involved lung can then be calculated. Fig. 4 presents data interpreted in this manner. After pneu-

monectomy, of course, the loss was 100 per cent. Losses in decreasing order were then found for pneumothorax, lobectomy, and thoracoplasty. Extrapleural pneumothorax caused the least loss of oxygen uptake while phrenic interruption caused the least loss of ventilation.

From bronchospirometric studies it was therefore possible to compare the loss of function after any particular procedure as applied to the same type of lung. Extrapleural pneumothorax was performed on lungs carrying over 40 per cent of the functional load in 18 of 27 patients. Fig. 5 presents a comparison of the losses of maximum breathing capacity and vital capacity of various operations on lungs which carried 40 per cent of the total oxygen uptake or more. There were few cases in our files of pneumonectomy done

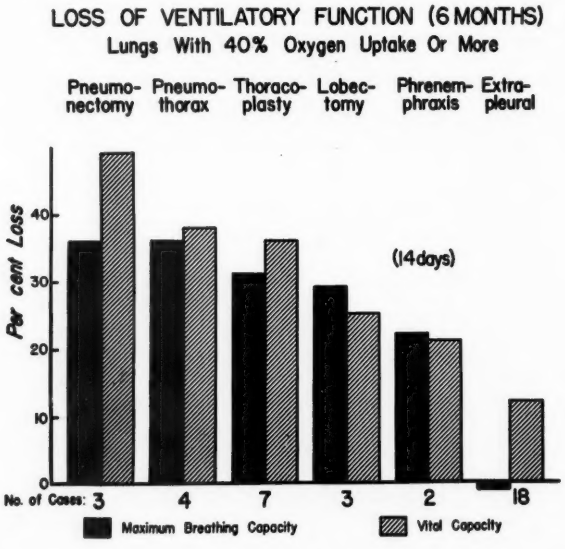

Fig. 5.—Per cent loss of maximum breathing capacity and vital capacity for various collapse procedures and resection performed on lungs which participated to the extent of 40 per cent or more in oxygen uptake before operation. Losses were calculated from six-month studies with the exception of phrenic interruption.

on such lungs. The only three illustrated were done for small malignant tumors. The same, to a lesser extent, applies to all other operations for 40 per cent lungs. Again in this group, pneumonectomy and pneumothorax led the decreasing order of functional losses. Thoracoplasty caused a poorer end result than lobectomy when compared in this fashion, a fact not apparent from the over-all charts of Figs. 2 and 4. The loss after phrenic interruption of such well-functioning lungs was also relatively great. Extrapleural pneumothorax caused the smallest decrease of vital capacity and resulted in a small gain of maximum breathing capacity.

Individual Cases.—One of the difficulties in the physiologic evaluation of any particular type of surgical procedure is the estimation of the effect on the

unoperated lung. A measured decrease in maximum breathing capacity or residual air, for example, may be due to the effect of collapse of the operated lung alone, it may be due to loss of function of both lungs, and it may be the end result of a combination of loss of function on the operated side and com-

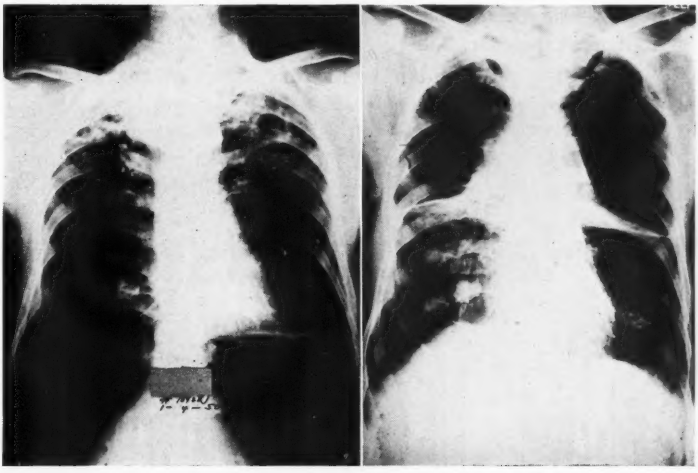

Fig. 6 (Patient No. 14-15).—Preoperative and postoperative posteroanterior roentgenograms of the chest.

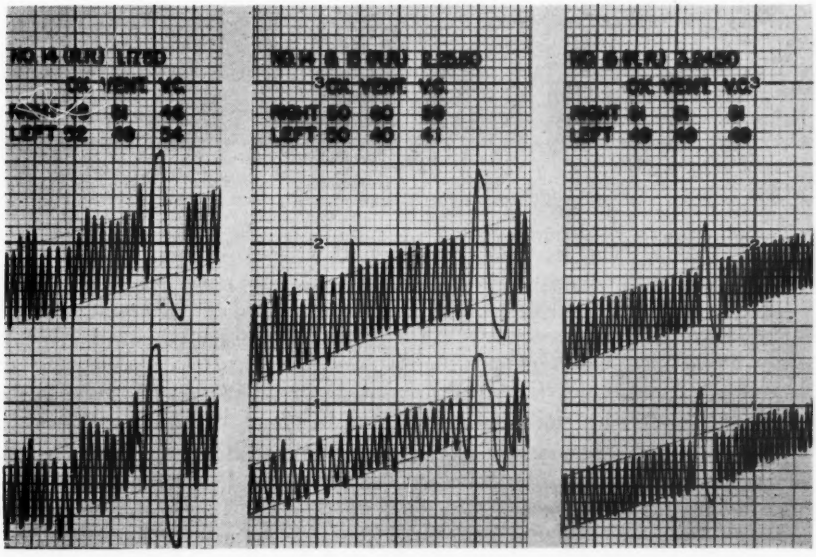

Fig. 7 (Patient No. 14-15).—Bronchospirometric tracings from left to right: before operation, after right extrapleural pneumothorax, and after bilateral extrapleural pneumothorax. Upper tracing: right lung.

pensatory gain on the other side. In the present series there were two patients on whom bilateral extrapleural pneumothorax was performed (Case 10-11 and Case 14-15); two patients where the contralateral lung was almost functionless (Cases 8 and 9); and last, one operation was performed on a remaining lung three years after contralateral pneumonectomy. No compensatory changes could occur here and the unique opportunity presented itself for study of the physiologic effect of the operation.

Fig. 6 shows the posteroanterior roentgenogram of the chest of patient No. 14-15 before and after bilateral extrapleural pneumothorax. Fig. 7 shows the bronchospirogram of the same patient before operation, after left extrapleural, and after both operations. The shift of function to the unoperated lung, unlike that after any other collapse procedure, here was hardly perceptible.

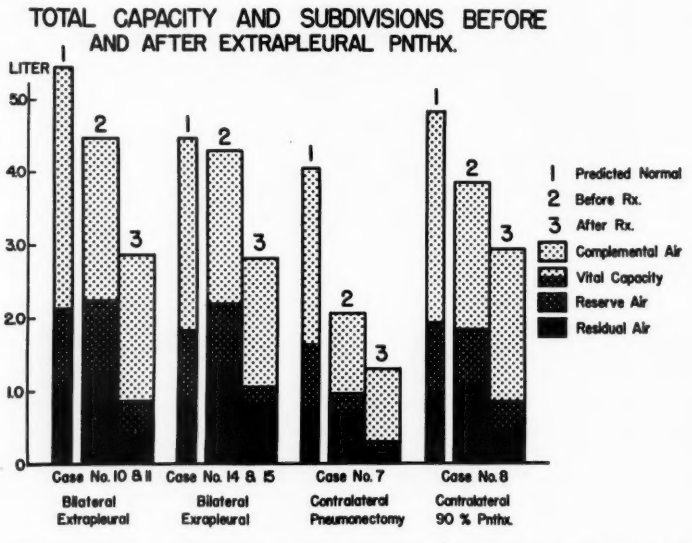

Fig. 8.—Predicted (*1*), preoperative (*2*) and postoperative (*3*) pulmonary total capacities and subdivisions. From left to right: Patients No. 10-11, No. 14-15, No. 7, and No. 8.

Fig. 8, section 2, shows the predicted total capacity and the determined values before and after both operations. The total capacity before operation was nearly normal but slight pulmonary emphysema was present. The ratio of $100 \times RA/TC$ was 32, larger than the normal ratio of 24.[27] The decrease in the total capacity after operation was chiefly due to loss of functional residual air.

Total capacities before and after the other bilateral extrapleural pneumothorax in Case 10-11 are illustrated in Fig. 8, column 1. This patient's postoperative chest roentgenogram is included in Fig. 13. Physiologic changes of lung volumes were much the same as in the previous case. The initial total capacity was somewhat more reduced and again reduction after both operations was chiefly due to decrease of residual and reserve airs.

Patient No. 8 (Fig. 8, column 4) shows a surprisingly large total capacity in view of the fact that one of the patient's lungs was almost completely destroyed. Changes in the subdivisions of the total capacity were again much the same as in the previous cases, only here there was no reduction whatever of complementary air. The postoperative residual air volumes for the three cases of 625, 415, and 480 c.c., respectively, are, to our knowledge, the lowest values ever recorded.

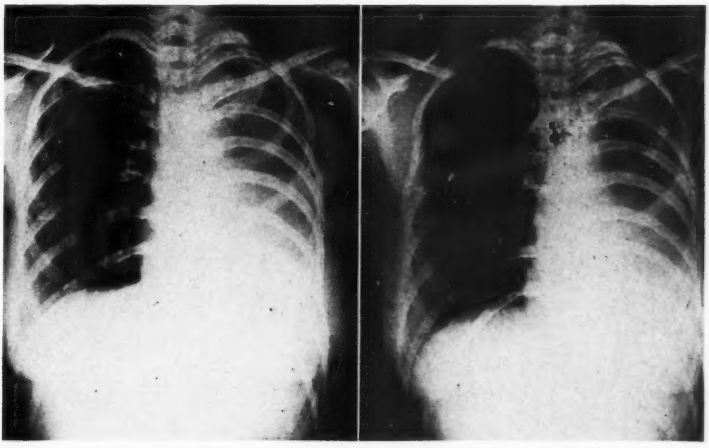

Fig. 9 (Patient No. 7).—Preoperative and postoperative posteroanterior roentgenograms of the chest.

The patient (Case 7) whose pre- and postoperative roentgenograms of the chest are recorded in Fig. 9 had had a left pneumonectomy three years prior to study with an operative spread to the contralateral apex. An oleothorax was induced on the left and the patient carried pneumoperitoneum for two years in an attempt to collapse a small right apical cavity and to control intermittent hemoptyses. A right extrapleural pneumonolysis with mediastinal dissection to the azygos vein was carried out eight months ago by one of us (J. W. S.) with sputum conversion. Gastric washings after suitable walking exercises have all been negative. This patient's maximum breathing capacity decreased from 65 to 56 liters per minute although from the appearance of the postoperative roentgenogram almost one-half the remaining lung appears collapsed. Since the exercise program was started, the patient has noted no dyspnea after rapid level walks and after climbing two flights of stairs. Her initial total capacity was one-half of normal (Fig. 8, column 3) with slight emphysema ($100 \times RA/TC$;35). Again the complementary air remained almost unchanged and the residual air decreased from 720 to 200 [*sic*] c.c.

The last patient (Case 9) had a right ten-rib thoracoplasty several years ago (Fig. 10). Left apical cavitary disease was complicated by advanced

pulmonary emphysema (100 × RA/TC:47) and he was slightly dyspneic even at rest. A small extrapleural pneumonolysis was performed in another institution.* Preoperative and postoperative bronchospirograms (Fig. 11) show no change in relative oxygen consumption. The patient's pulmonary mixing problem was improved since the 100 × RA/TC decreased from 47 to 37 and the complementary air was actually increased after operation because the residual air decreased more than the vital capacity.

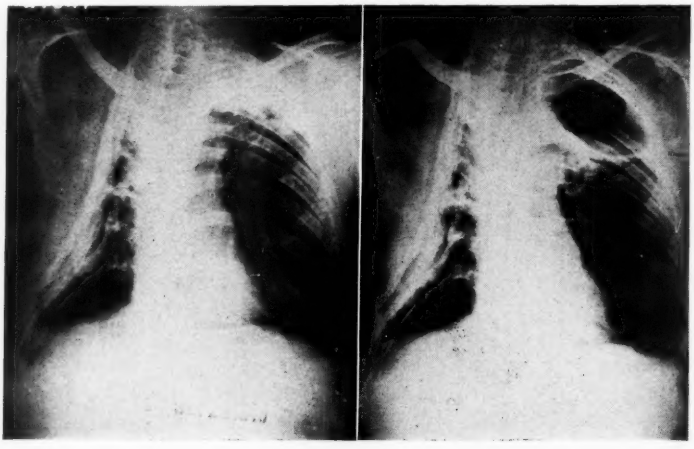

Fig. 10 (Patient No. 9).—Preoperative and postoperative posteroanterior roentgenograms of the chest.

Group 2

All three patients in this group had very limited respiratory reserve even before operation (Tables I and II). Two of the three showed over 70 per cent of the total function on the side under consideration for surgery (Table III). One of them (Case 27) had had a previous partially successful decortication of an unexpandable pneumothorax lung on the contralateral side and the other (Case 28) had an old four-rib thoracoplasty. In all three cases the pleura was accidentally opened at operation and intrapleural hemopneumothorax persisted for over fourteen days after operation in spite of assiduous and repeated aspirations. The postoperative functional picture here was entirely different from patients of Group 1. The mean vital capacity decreased from 1.94 to 0.82 liters fourteen days after operation, a loss of 58 per cent. The mean maximum breathing capacity decreased from 61 to 31 liters per minute, or 49 per cent. Two of the three patients (Cases 27 and 28) were severely dyspneic postoperatively and both remained on oxygen therapy for over two weeks.

A summary of the effect of parietal pleura tear at operation is given in Fig. 12. In 22 cases where this accident did not occur, the losses for vital

*Cambridge Sanatorium, Dr. Francis Woods.

capacity and maximum breathing capacity fourteen days after operation were 14 and 4 per cent, respectively. In 4 cases where the pleura was opened but intrapleural pneumothorax was avoided by intrapleural catheter drainage and underwater seal these losses were 23 and 14 per cent, respectively, while in the 3 cases just discussed the losses were 58 and 49 per cent.

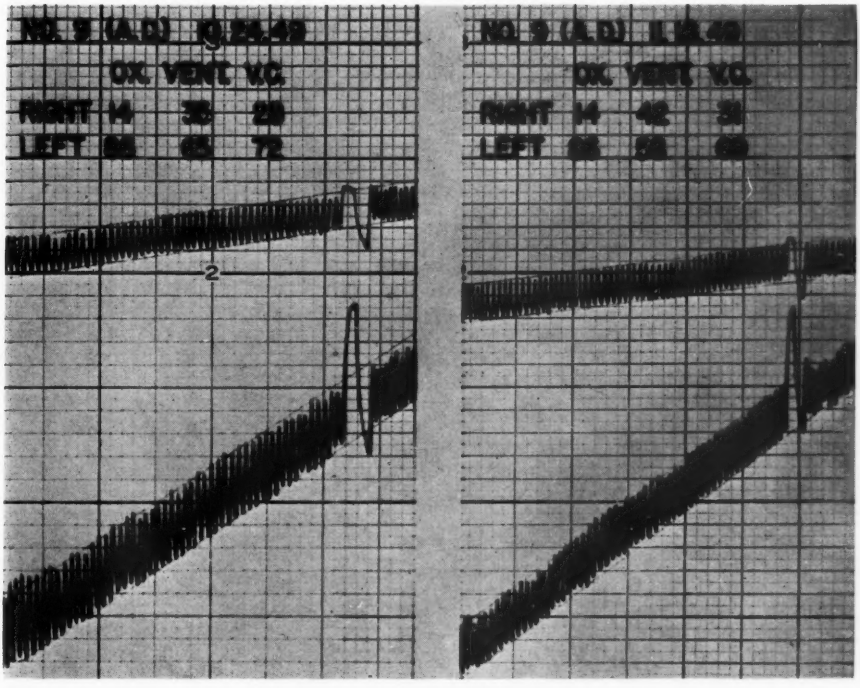

Fig. 11 (Patient No. 9).—Preoperative and postoperative bronchospirometric tracings. Upper tracing: right lung.

DISCUSSION

The aim of collapse therapy is to provide maximal collapse for diseased tissue with as little interference as possible with physiologic function of healthy lung tissue. Extrapleural pneumothorax conforms more closely to these aims than any other form of collapse therapy. There is minimal interference with ventilatory function as expressed by postoperative maximum breathing capacity. Collapse takes place almost exclusively at the expense of functional residual air.

It now remains to discuss the reasons for these striking functional end results. Following most forms of resection or collapse therapy losses of pulmonary function are encountered which are not due to collape per se but are due to a variety of undesirable physiologic alterations of the function of the chest wall, the diaphragm and mediastinum, and the pleura. Table IV presents an attempt at summarization of these effects for various types of collapse procedures.

TABLE IV. LOSS OF PULMONARY FUNCTION DUE TO FACTORS OTHER THAN COLLAPSE

	"MODERN" THORACOPLASTY	SUBCOSTAL PLOMBAGE	PULMONARY RESECTION	PHRENIC INTERRUPTION	INTRAPLEURAL PNEUMOTHORAX	PNEUMOPERITONEUM	EXTRAPLEURAL PNEUMOTHORAX
1. *Postoperative Period*							
Paradoxical motion of chest wall	++	+	0	0	0	0	0
Mediastinal swinging motion	++	+	0	+	+	0	0
Postoperative pain	++	++	+	0	0	±	±
2. *Persistent Changes*							
Interference with intrapleural pressure mechanism	0	0	0	0	++	0	0
Destruction of rib cage	++	++	0	0	0	0	0
Loss of diaphragmatic motion	+	+	+	++	0	+	0
Paradoxical motion of diaphragm	0	0	0	++	0	0	0
Compensatory increase of residual air (emphysema)	+	+	++	0	0	0	0
Residual pleural thickening and parietal fixation of lung	±	±	++	0	++	0	0

A number of factors are at play during the immediate postoperative period which are responsible for a greater pulmonary insufficiency as measured fourteen days after operation than six months after operation. These factors are chiefly pain due to interruption or traumatization of intercostal and other nerves, and paradoxical motion of the decostalized portion of the chest wall. Both of these factors are chiefly responsible for the great loss of function seen fourteen days after thoracoplasty (Fig. 1). Whether actual paradoxical motion of the air from one lung to the other (Pendelluft) also occurs with staged thoracoplasty is a matter of considerable debate. Subcostal, extraperiosteal plombage presents one of the many attempts of avoiding paradox seen after standard thoracoplasty.

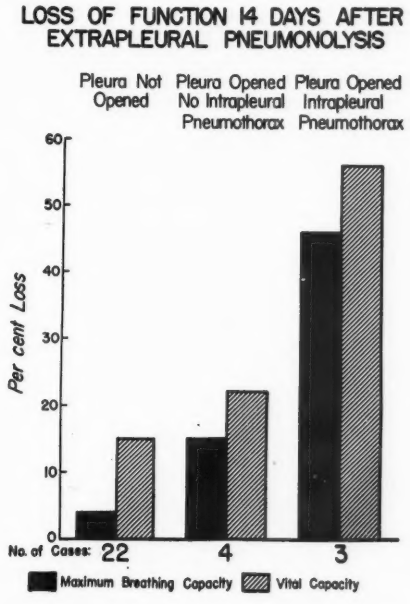

Fig. 12.—Per cent loss of maximum breathing capacity and vital capacity 14 days after extrapleural pneumothorax. From left to right: 22 patients in whom pleura was not opened during operation, 4 patients in whom pleura was opened but intrapleural pneumothorax was avoided, and 3 patients in whom pleura was opened resulting in marked hemopneumothorax.

In our experience this purpose is only partially accomplished. Pain after extrapleural pneumonolysis is usually minimal. Only a small segment of rib is removed, the intercostal nerve is left intact and the use of retractors and rib spreaders is avoided. Mediastinal swing after thoracoplasty is part of the paradox syndrome and is to some extent responsible for impaired function of both lungs. Its role after phrenic interruption has been previously discussed by one of us (E. A. G.). Suffice it to say here that the mediastinum can swing sufficiently under certain conditions to cause normal ventilation of a lung which is not otherwise caused to move either by the hemidiphragm or the thoracic rib cage.[26]

There are a number of factors other than collapse responsible for persistent loss of function during the entire period the collapse is maintained (Table IV). The most important of these is interference with the intrapleural pressure mechanism. As long as the intrapleural space is merely a "potential" one, the surface of the lung must follow completely every motion of the diaphragm and rib cage. If only a small rim of intrapleural pneumothorax is created, a damping effect occurs which can be well visualized on fluoroscopy. The lung still follows the motion of the thoracic cage but only in a general way, its margin or surface moves less than the chest wall. An additional deleterious effect on the contralateral lung no doubt takes place due to mediastinal swing and altered intrathoracic pressure relationships. The end result of all these physiologic alterations on function can be seen not only from the various charts of comparative functional loss presented (Figs. 2 to 5), but are also evident from the changes which take place after uncomplicated extrapleural pneumothorax and after extrapleural pneumonolysis complicated by accidental intrapleural hemopneumothorax (Fig. 12).

Destruction of a portion of the thoracic cage, as encountered in thoracoplasty or extraperiosteal plombage, is deleterious to pulmonary function from three different aspects. The motion of the rib cage on the operated side is destroyed to a great extent even if the lower six or seven ribs are left intact, because the orderly sequence of the nervous mechanism of respiration is interrupted. The leaf of the diaphragm on the operated side becomes less efficient in its function since its full relaxation on expiration is ordinarily aided by a simultaneous contraction of the chest wall which exerts an infolding effect on the diaphragm.[28] A reduction in range of motion of the hemidiaphragm after thoracoplasty is always noted on postoperative fluoroscopy.[25] The third effect of a partially destroyed thoracic cage was recently pointed out by Cournand and associates.[29] The scoliosis due to thoracoplasty places the contralateral lung also at a relative mechanical disadvantage. Loss of diaphragmatic motion after pneumoperitoneum is never complete but occasionally quite marked. A decreased extent of motion is frequently found after lobectomy and after pneumothorax if the latter is complicated by hydrothorax. "Compensatory emphysema" of the remaining lobes after lobectomy and of the contralateral lung after pneumonectomy may play a role in the loss of function, particularly the respiratory component. This factor has been investigated for some years but a final answer to the problem is still to come.

From a comparison of the vertical columns of Table IV it is apparent that the physiologic disturbances enumerated previously involve extrapleural pneumothorax to a lesser extent than any other form of resection or collapse procedure used in the treatment of pulmonary tuberculosis. Fig. 13 shows postoperative roentgenograms of the chest of three of our patients in deep inspiratory and expiratory position. Preservation of diaphragmatic and rib cage motion is well illustrated. The extrapleural air space under positive pressure acts as a semisolid body which transmits the motion of the entire thoracic cage to the partially collapsed lung with the result that the maximal

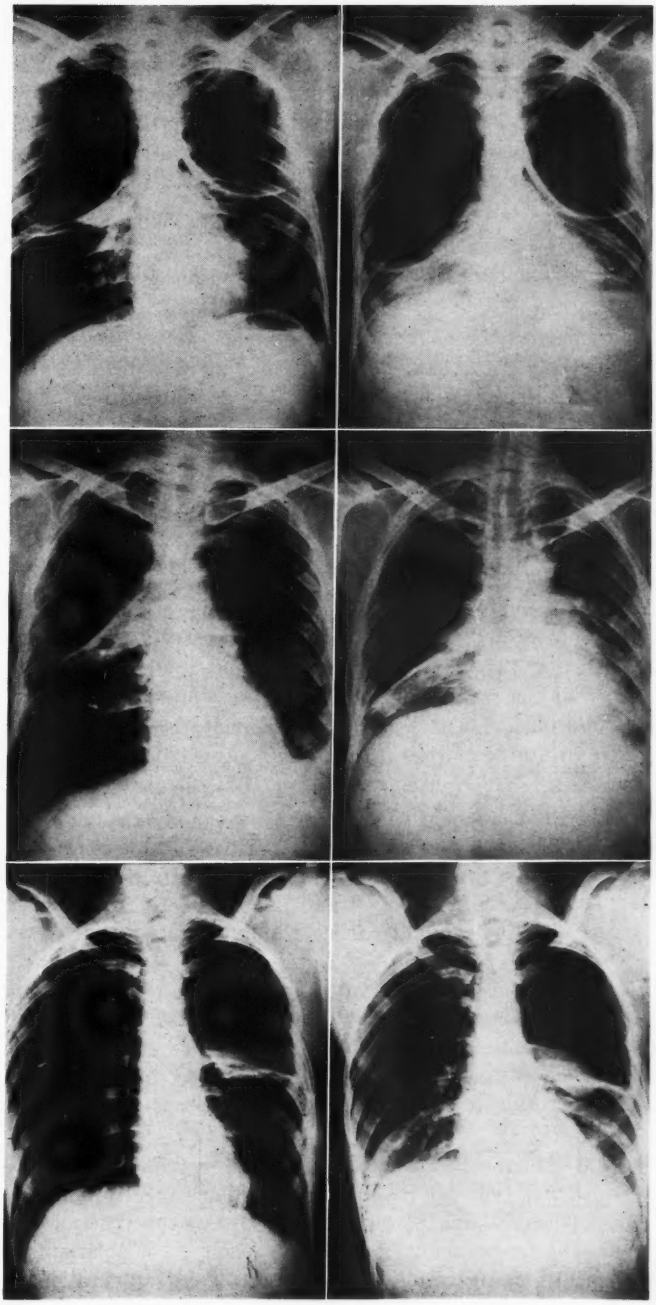

Fig. 13.—Posteroanterior roentgenograms of the chest on deep inspiration (left) and fullest expiration (right). Patients No. 10-11 (top of page), No. 13 (middle of page), and No. 26 (bottom of page).

air exchange remains unchanged and the extrapleural space is in effect subtracted from the residual air on expiration. The lungs in full expiration are almost devoid of air and in the roentgenogram have an almost atelectatic appearance. The floor of the extrapleural space can be seen to move several interspaces. This is best visualized on a kymogram.[6]

The authors are fully cognizant that the factors which place extrapleural pneumothorax in a favorable light functionally are the very factors which do not permit the desirable pulmonary "rest" in the sense of functional elimination.

Without entering the controversy concerning the indications for extrapleural pneumothorax, the data presented suggest that this operation can be carried out with the certain knowledge that the resulting loss of pulmonary function will be very small or nonexistent. The operation can, therefore, be offered to a number of patients whose pulmonary reserve would not permit any other type of collapse therapy. It is our hope that during the present re-evaluation of extrapleural pneumothorax both as a definitive procedure and as a preliminary to thoracoplasty these studies will aid in placing this operation in its proper light.

Although one cannot be other than enthusiastic solely on the basis of the comparatively favorable functional response to extrapleural pneumothorax, it is recognized that this procedure in itself is not a substitute at the present time for many of the proved collapse measures. Much time must elapse in order that opportunity shall be afforded the natural history of pulmonary tuberculosis to bring the end results into their proper perspective as it always has in any procedure which has been used to alter the course of any disease.

However, if the passage of time should show that single-stage extrapleural pneumothorax, freed of its complications by the use of biotherapeutic agents, offers at least as much to the patient as modern multiple stage thoracoplasty without comparable loss of function and deformity, the operation may well find its place as a leading contender in the choice of procedures.

SUMMARY

1. Loss of pulmonary function was studied by maximum breathing capacity, vital capacity, residual air, ventilation on exercise, breathing reserve, and bronchospirometry before operation and fourteen days and six months after operation of extrapleural pneumothorax on 30 occasions on 28 patients.

2. The loss fourteen days after operation was 18 per cent for vital capacity and 5 per cent for maximum breathing capacity, and the loss six months after operation was 12 per cent for vital capacity and there was a gain of 2 per cent for maximum breathing capacity. In 3 patients where the pleura was opened at operation with a resulting intrapleural hemopneumothorax the losses fourteen days after operation were 58 per cent for vital capacity and 49 per cent for maximum breathing capacity.

3. Bronchospirometric studies before operation showed an average function of 56 per cent of the total for the side to be operated upon since 83 per cent of the patients had contralateral collapse therapy or cavitary disease before operation. After operation there was a 5 per cent shift of oxygen uptake and an 8 per cent shift of ventilation and vital capacity to the contralateral lung.

4. Loss of function after extrapleural pneumothorax was compared to losses which occured after 121 thoracoplasties, 24 pneumonectomies, 20 lobectomies, 16 intrapleural pneumothoraces, and 9 phrenic interruptions. Both the total losses as calculated from maximum breathing capacity and vital capacity and the shift of function as calculated from bronchospirometry were smaller after extrapleural pneumothorax than for any other collapse procedure or resection studied. This was even more evident when operative losses were compared as performed on lungs with the same extent of preoperative function as determined by bronchospirometry.

5. Decrease of total pulmonary capacity after extrapleural pneumothorax took place chiefly at the expense of functional residual air.

6. Causes of loss of function other than that due to collapse for various operative procedures are discussed.

The authors take pleasure in acknowledging their indebtedness to Miss Dorothy M. Donnelly for her technical assistance.

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DISCUSSION

DR. VIKING OLOV BJÖRK, Stockholm, Sweden.—In the beginning of 1930, bronchospirometry was introduced by Jacobaeus, Frenckner, and Björkman. Since then the main difficulty has been to obtain large enough inner diameter of the tubes to prevent stenotic breathing and achieve reliable values. Pulmonary function studies at the Sabbatsberg Hospital in Stockholm have therefore been of practical value only since Carlens designed his double-lumen catheter.

(Slide.) You see the double-lumen catheter in place; it is provided with a small hook that is automatically engaged by the carina. This rubber hook is tied down to the catheter with a silk thread until it has been passed through the larynx, when the slipknot is untied. When the catheter is pushed down it automatically enters the left main bronchus. It is then easy to feel when the hook is engaged by the carina. Neither fluoroscopy nor bronchoscopy is necessary. Then the cuffs are inflated. The hook and the two rubber cuffs will maintain the catheter in the correct position. It cannot occlude the left upper lobe bronchus. The inner diameter is 7 mm. so that no stenosis is produced. Thus, the same value of the total capacity and its different fractions, both at rest and during exercise tests, are obtained when the values from the two lungs using the double-lumen catheter are added, as compared with the values obtained in the usual way with a mouthpiece. Also, the values of maximum breathing capacity are the same with the tube in place as without the tube.

It is as easy to introduce this catheter as an ordinary intratracheal tube for anesthesia, and it is now being used in the Crafoord Clinic as an ordinary anesthesia tube in cases with risk of spillover during operation.

(Slide.) This shows one lung anesthesia during lobectomy of a right upper lobe for an abscess. Continuous suction is applied.

(Slide.) When the bronchus is dissected and clamped, the rest of the right lung is connected with the spiropulsator and ventilated. This method is preferred to the face-down position.

DR. STRIEDER (closing).—I would simply like to say, in view of some comment in the corridors relative to extrapleural pneumothorax, that we do not propose it, as has been advocated in the past, as an operative panacea for tuberculosis. As Dr. Gaensler indicated, however, it is an operation which can be done when other operations cannot be done, and certainly in the experience of us all there are many such cases where the borderline residual function is such that a major procedure would certainly tip the patient over and make him a respiratory cripple, if he survived.

(Slide.) I would like briefly to show two such cases. The first is a 24-year-old white man who had pneumoperitoneum and bilateral cavitary disease for four years. The pneumoperitoneum was held until the second stage of bilateral extrapleural pneumothorax was completed.

(Slide.) This shows function studies before and after the right side, and after both sides, with a loss of some 7 liters of maximum breathing capacity. Vital capacity dropped from 3.2 to 2.4 liters, and breathing residual dropping only one per cent.

(Slide.) The second patient is a 24-year-old girl who had a total pneumonectomy on the left for a destroyed lung, some three years prior to her extrapleural pneumothorax. In the ensuing years she had developed the apical cavity which you see on the right and had intermittent hemoptysis with positive sputum. She also had a tuberculous empyema on the left which apparently had been controlled over the three-year period with oleothorax.

(Slide.) The maximum breathing capacity, before extrapleural pneumothorax, was 65 liters per minute, and it dropped only 9 liters, to 56, after extrapleural pneumothorax. The breathing reserve dropped from 68 cent to 62 per cent and, believe it or not, residual air was only 200 c.c. This girl is not dyspneic on mild physiologic exercise; she is walking comfortably on the level.

PULMONARY TUBERCULOMAS

Pathogenesis, Diagnosis, and Management

Gordon J. Culver, M.D. (by invitation), Joseph P. Concannon, M.D. (by invitation), and Joseph E. MacManus, M.D.
Buffalo, N. Y.

THROUGH the increasing use of routine roentgen examinations of the chest, many solitary pulmonary masses have been discovered. These include both malignant and benign lesions. Tuberculomas represent the largest single group of these benign lesions. Tuberculosis of this type is rather loosely classified in the literature. There has been no complete statement clarifying the pathogenesis of this tuberculous process. Certainly no single explanation is adequate for all cases, and in any series the pathogenesis will vary from case to case.

Extrapulmonary tuberculomas have been adequately described. There have been, however, some inaccuracies in the use of the term, *pulmonary tuberculoma*. A tuberculoma is merely a tumorlike granuloma caused by the tubercle bacillus, encapsulated by connective tissue, and showing no evidence of surrounding inflammation or spread. These "tumors" may be completely or partially calcified, and may have many scattered caseating areas within the mass. They are usually oval or round in shape, and may vary from the size of a bean to the size of an orange, or larger. The term *tuberculoma* should probably be restricted to the tuberculous granuloma having a diameter of at least 2 cm. This is admittedly an arbitrary classification, but it serves to prevent further confusion in the literature as to the use of the term.

We believe that a pulmonary tuberculoma may arise in several ways:[12] (1) the encapsulation of a giant primary focus—this focus may occur at any age; (2) the encapsulation of a restricted reinfection focus of tuberculosis that has a tendency toward rapid regression, and that becomes homogeneous; (3) the completely blocked tuberculous cavity with resultant inspissation of caseous material, fibrosis, calcification, and shrinkage of the occluded cavity; this cavity may occur in the reinfection focus, and rarely in the Ghon focus.

Many theories have been advanced describing the mechanism responsible for the closure of tuberculous cavities, and undoubtedly cavities heal by different mechanisms. Coryllos has drawn attention to the closure of tuberculous cavities by means of bronchial occlusion. Auerbach and Green presented four cases in which closure and healing of tuberculous cavities occurred by bronchial occlusion. Loesch, in 1944, presented a detailed pathologic description of complete closure of cavities, and his pathologic findings are in close accord with those of Coryllos. The occlusion of a stem bronchus to a cavity may result in the formation of a tuberculoma. The bronchi which are tributary to the draining

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Read before the Thirtieth Annual Meeting of The American Association for Thoracic Surgery, Denver, Colo., April 15-18, 1950.

cavities are almost always involved by the tuberculous process to varying degrees of severity. This tuberculous process in the bronchi produces mechanical changes, chiefly narrowing or complete occlusion of the lumen. With a patent unnarrowed bronchus, there is a free passage of air to and from the cavity on respiration. As is well known, tension cavities often form with incomplete bronchostenosis. With complete occlusion of the bronchus there is gradual absorption of air within the cavity, and atelectasis in the area of lung supplied by the bronchus. The anoxemia of the atelactatic lung which suppresses ventilation and circulation will cause fibrosis. Eventually the wall of the cavity will be completely replaced by a fibrous tissue. The cavity may now fill in with caseous material, and be visible on the roentgenogram as a solitary circumscribed shadow. The fate of the erstwhile cavity is now uncertain. Pinner stated: "Cavities that become blocked by bronchial occlusion may sometimes, after long periods of blockage, reopen and produce bacilliferous sputum and intrabronchial dissemination." He presented roentgenograms of a case illustrating the reopening of a tuberculous cavity. On the other hand, regressive changes may occur, the caseous material becoming inspissated and perhaps calcified. Actual bone formation has been described in tuberculomas. Case 1 illustrates the method by which a bronchial communicating cavity becomes a solid tumor, most likely by means of a bronchial occlusion.

This paper is primarily concerned with tuberculomas within the pulmonary parenchyma; however, tuberculomas may occur within the mediastinum. The formation of a mediastinal tuberculoma is due to a caseating process in the mediastinal lymph nodes. Six cases of mediastinal tuberculoma have been reported in the literature.[13, 14, 25] Case 10, in the opinion of the operating surgeon, was a mediastinal tuberculoma, although pathologic evidence is lacking. Case 12 fits all the criteria for a diagnosis of mediastinal tuberculoma.

Tuberculomas have been described as occurring in the central nervous system, brain, orbit, middle ear, breast, stomach, nasal septum, paranasal sinuses, and abdominal wall and lungs. Tuberculomas of the pulmonary fields are not infrequent. In 1931, Albert described round foci which he believed represented encapsulated tuberculous lesions. In 1932, Straub observed eight cases of single and multiple round tuberculous foci. He observed that some of these would undergo changes only after a long period of time. Klein and Wolff, in 1934, added six more cases to the literature, five of which showed inactivity of the round foci after one or two years. One case which came to autopsy revealed a lesion which had remained stationary for about six months, only to break down and be the cause of a spreading exudative tuberculosis. At autopsy the characteristic lesion consisted of a chalky caseous substance encircled by more centrally deposited loose connective tissue and a peripheral dense hyaline connective tissue capsule. Birkelo and Kasper, in 1939, described the radiographic appearance and progress of the circular lesions of tuberculosis. Many of these lesions were stable and could be classified as tuberculomas. Maurer-Mast and Franklin, in 1941, reported a case of tuberculoma, and stated that there were only two cases of similar nature reported in the literature. How-

ever, many of the solitary round foci of tuberculosis described in the literature previous to this report were undoubtedly tuberculomas. Jones and Dolley, and Dieffenbach, in 1940, each reported a case of tuberculoma for which a total pneumonectomy had been done because of a mistaken diagnosis of bronchiogenic carcinoma. Since that time many tuberculomas have been described, notably in the surgical literature.[9, 10, 11, 26] Surgery, in many of these cases, was undertaken with a mistaken diagnosis of lung tumor. Mahon and his associates had in their series 35 patients treated by pulmonary resection, either lobectomy or wedge-shaped excision for tuberculoma.

The roentgenographic diagnosis of solitary shadows within the lung fields is frequently extremely difficult. While the roentgenographic visualization of the chest is invaluable for demonstrating the presence of a lesion, it cannot always be relied upon for an adequate differentiation. It is beyond the intent and scope of this paper to describe the complete roentgenologic findings of various tumors found within the lung fields.

The differential diagnosis of circumscribed solitary shadows within the lung fields must always include both primary and secondary neoplasms, tuberculomas, encapsulated effusions, cysts, abscesses, localized areas of pneumonitis, hamartoma of the lung, arteriovenous aneurysms, and other more rare lesions. Occasionally a neoplasm in the mediastinum will be drawn out into the lung fields by negative intrathoracic pressure. These tumors are usually benign, because malignant mediastinal tumors are fixed and remain within the mediastinum. Many of these lesions are uncommon, and others can be excluded by special x-ray techniques.

The solitary circumscribed shadow of the tuberculoma is readily confused with the peripheral type of pulmonary carcinoma. The two lesions, however, may occasionally be readily differentiated. The presence of calcification, to our knowledge, has never been observed roentgenographically within primary malignant tumors. The type of calcification seen radiographically in tuberculomas is variable. A tuberculoma may be so completely calcified that it resembles a giant mulberry. The calcification may be arranged as a single concentric ring at the periphery of the tumor, or in the form of multiple concentric rings. There may be discrete scattered areas of calcification throughout the tumor. Calcification which is not seen in ordinary projections of the chest may be visualized by tomographic studies. Other radiographic features which suggest the diagnosis of tuberculoma are multiple areas of diminished density throughout the tuberculoma which represent areas of caseation and adjacent areas of calcification and fibrosis. The tuberculoma is oval, round, or lobulated, and regular in outline. Rarely there may be multiple tuberculomas. Tuberculomas are often seen which are homogeneous in density, and do not contain any calcium. It will occasionally be noticed that this type of tuberculoma may enlarge when observed over a short period of time. It is the calcium-free tuberculomas which cannot be differentiated roentgenographically from bronchiogenic carcinoma of the peripheral type.

A bronchiogenic carcinoma may have a smooth, round contour and lie in the peripheral lung fields. It may have a homogeneous density, or it may

have areas of diminished density from breakdown throughout the tumor. This type of bronchiogenic tumor apparently tends to grow by expansion rather than infiltration. However, it has been our experience that primary peripheral neoplasms in most instances show definite roentgen evidences of infiltration beyond the main mass of density. Completely encapsulated, noninfiltrative primary neoplasms are extremely rare as compared with tuberculomas and other benign lesions.

Tumors of the prostate, testicle, and kidney tend to metastasize to the lungs, and occasionally appear as solitary circumscribed lesions within the lung fields. Usually, however, within a few weeks multiple nodules appear in the lung fields and simplify the diagnosis. Thornton, Adams, and Bloch state that, although they see thirty multiple metastatic tumors per year, they have seen only three solitary metastases in ten years. There is no way of differentiating the slowly-growing metastatic lesion from a bronchiogenic carcinoma, except to search for a primary lesion. Rarely, multiple tuberculomas may occur in the lung fields and be mistaken for metastases to the lung. Dahl-Iverson and Møller reported a case in which two tuberculomas were present within the pulmonary parenchyma in the lower right lobe, one posterior and one anterior. We have seen one case in which two tuberculomas were present, for which lobectomy was done. Fortunately, these lesions were calcified, and a diagnosis of tuberculoma was made preoperatively. We have also seen another case in which a radiographic diagnosis of double tuberculoma was made. However, this case did not come to operation.

The greatest percentage of solitary peripheral masses, both with and without surrounding infiltration, will prove to be carcinoma of the peripheral type, which type constitutes approximately 25 per cent of all primary lung tumors. Thornton, Adams, and Bloch presented twenty-three cases of solitary "circumscribed tumors" which had been observed at the University of Chicago clinics in a ten-year period. Seventeen of these were malignant lung tumors, fourteen primary, three were metastatic. Six were benign, one hemangioma, four tuberculomas, one a chronic lung abscess. Since the majority of the solitary peripheral tumors are due to malignant disease, we must consider every lesion of this type malignant until proved otherwise, or we shall continue to fall into the errors of the past. Patients with bronchiogenic carcinoma are occasionally admitted to tuberculosis sanatoriums and the malignant process is far advanced before the correct diagnosis is made. Pillsbury and Wassersug reported on ten patients who were admitted to a tuberculosis sanatorium in a ten-year period, who were later proved to have had bronchiogenic carcinoma.

It is our belief that a more extensive and complete roentgenologic study would result in an almost absolute roentgen diagnosis of malignant neoplasm in many instances, for which immediate definitive therapy could be carried out.

The problem of deciding the best treatment for a patient who has been referred to a surgeon, with an asymptomatic, solitary, well-circumscribed mass in the lung field, is not always an easy one, and every weapon in our diagnostic armamentarium must be utilized. The patient should be fluoroscoped and

roentgenograms taken. Tomography may be of value in demonstrating areas of calcification. Efforts should be made by the medical consultant to rule out primary neoplastic disease of other organs, particularly kidney, prostate, or testicle.

Bronchoscopy may have been completed without gaining further information or help, particularly when the tumor is inaccessible to the bronchoscope, as in a tumor located in a peripheral bronchus. Secretions obtained bronchoscopically and stained by the Papanicolaou technique may result in a high percentage of positive diagnoses. Herbut and Clerf reported thirty cases of primary pulmonary carcinoma in which the bronchial secretions were stained by the Papanicolaou technique. Cancer cells were demonstrated in twenty-two cases, or 73 per cent. Studies done on these bronchial secretions may result in the demonstration of tubercle bacilli. A tuberculin test should always be done. If enlarged peripheral nodes are detected, they should be biopsied. Intrapleural fluid, if present, should be aspirated and cytologic studies carried out.

Tuberculomas in the pulmonary fields may be classified roentgenographically into two general groups. This grouping is important because it will be of great value in determining the activity of the lesion, and will indicate the necessity and the urgency of surgery.

The first group is *tuberculomas without calcification.* These tumors may be homogeneous in density, or may show one or more central areas of diminished density. Sputum studies may be negative or positive for tubercle bacilli. Some of these cases, through mismanagement, may have been followed for periods of six months or more without roentgenographic change in the size or character of the lesion. These tuberculomas constitute a threat to the continued well-being of the patient, because of the danger of imminent spread of the tuberculous process to the surrounding lung tissues. Case 2 illustrates the spread of a tuberculous process which could have been prevented by the early surgical extirpation of the tuberculoma. It must be noted in cases with a peripheral lesion of this type that the possibility of a bronchiogenic carcinoma has not been definitely ruled out, although there has been no roentgenographic progression of the lesion. It is for these reasons that immediate surgery should be recommended for the solitary round tumor without calcification, with or without areas of central breakdown.

The second group is *tuberculomas with calcification.* This group may be further divided into subgroups, depending on whether or not they contain central areas of breakdown. The calcified tuberculomas with radiographic areas of central radiolucency may be the focus of a spreading tuberculous process, and surgery may be recommended for these cases. Asymptomatic patients who demonstrate well-calcified tuberculomas in the lung fields, without central areas of radiolucency and with negative sputa, are in little or no danger of having a spread of this tuberculous process, so surgery is not recommended; instead, the patient is followed for long periods of time by serial roentgenograms. In fact, surgical intervention may be the cause of tuberculous complications. Case 7 illustrates some of the complications which may occur following the removal of a calcified tuberculoma.

CASE REPORTS

CASE 1.—M. A. was a 74-year-old woman who had numerous hospital admissions for bronchial asthma during the past fifteen years. She had low-grade temperature and a productive cough, occasionally with blood-streaked sputum, during these asthmatic episodes. She had massive hemoptysis in 1943, and again in April, 1948.

In April, 1942, radiographic examination of the dorsal spine showed a sharply demarcated cystic area in the right upper lobe, posteriorly. This cystic area measured 7 by 5 by 6 cm. In August, 1943, roentgenographic examination of the chest at the bedside showed a triangular area of density extending outward from the upper right hilar area, with gradually diminishing density at the periphery of the chest. This change was believed to be due to infection surrounding and obliterating the cystic lesion. One week later there was some surrounding pneumonitis, and the cystic nature of the lesion was again recognizable. Re-examination at the bedside in October, 1943, showed no appreciable change in the lesion. The next examination was in June, 1947, and there was now a rounded density in the area where the cyst was previously noted. There was no appreciable surrounding inflammatory reaction in the pulmonary tissue. In April, 1948, the cystic area was almost completely filled by dense material. The only aerated portion of the cystic area which remained was superiorly and posteriorly. The lesion was sharply demarcated and showed no surrounding infiltration, with no demonstrable calcification. The last roentgenographic examination of the chest, in May, 1949, showed no appreciable change in this lesion since examination of 1948.

Smears, cultures, and guinea pig inoculations were constantly negative for tubercle bacilli. Bronchoscopic examination was not done.

Discussion.—This case illustrates one of the methods of tuberculoma formation, and shows the transformation of a tension air-filled cavity to an almost solid tumor. This was probably due to the progression from partial to complete bronchial occlusion.

CASE 2.—G. H., a 32-year-old man, was examined roentgenographically by a mobile chest unit in April, 1946. A large circular area of increased density was noted at the level of the first interspace and second rib anteriorly on the right. There was no calcification in the mass. The remainder of the lung fields appeared clear. The patient had no symptoms or physical findings at that time. The diagnosis was undetermined, and interval studies directed.

The patient returned to the chest clinic in July, 1947, for a repeat roentgenogram, which showed no change since first examination. The impression at that time was that this lesion was most likely a tuberculoma. In April, 1948, he complained of weakness and fatigue. A roentgenogram of the chest was made, which showed a diffuse exudative lesion involving the greater portion of the right upper lobe. This had the appearance of a moderately advanced pulmonary tuberculosis with cavitation. Re-examination on June 23, 1948, showed further increase in the infiltrative lesions in the right upper lobe.

Sputum studies showed tubercle bacilli on smear and culture. The patient was sent to a tuberculosis sanatorium.

Discussion.—This case demonstrates the lurking malevolence of pulmonary tuberculomas. The possibility of either a malignant tumor or a tuberculoma should have been considered. In a tuberculoma without calcification the danger of spread into contiguous lung tissue cannot be over-emphasized. A thoracotomy should have been done in 1946 when this mass was first discovered.

CASE 3.—A. S., a 22-year-old white man, in February, 1946, was discovered to have a "spot on his lung." He was hospitalized for about four and one-half months at another institution, without any change in the size of the shadow. On discharge he had some shortness of breath and pain over the lower ribs on the right anteriorly. In January, 1947, he developed a productive cough, and had blood-streaked sputum on two occasions. Physical examination was essentially negative.

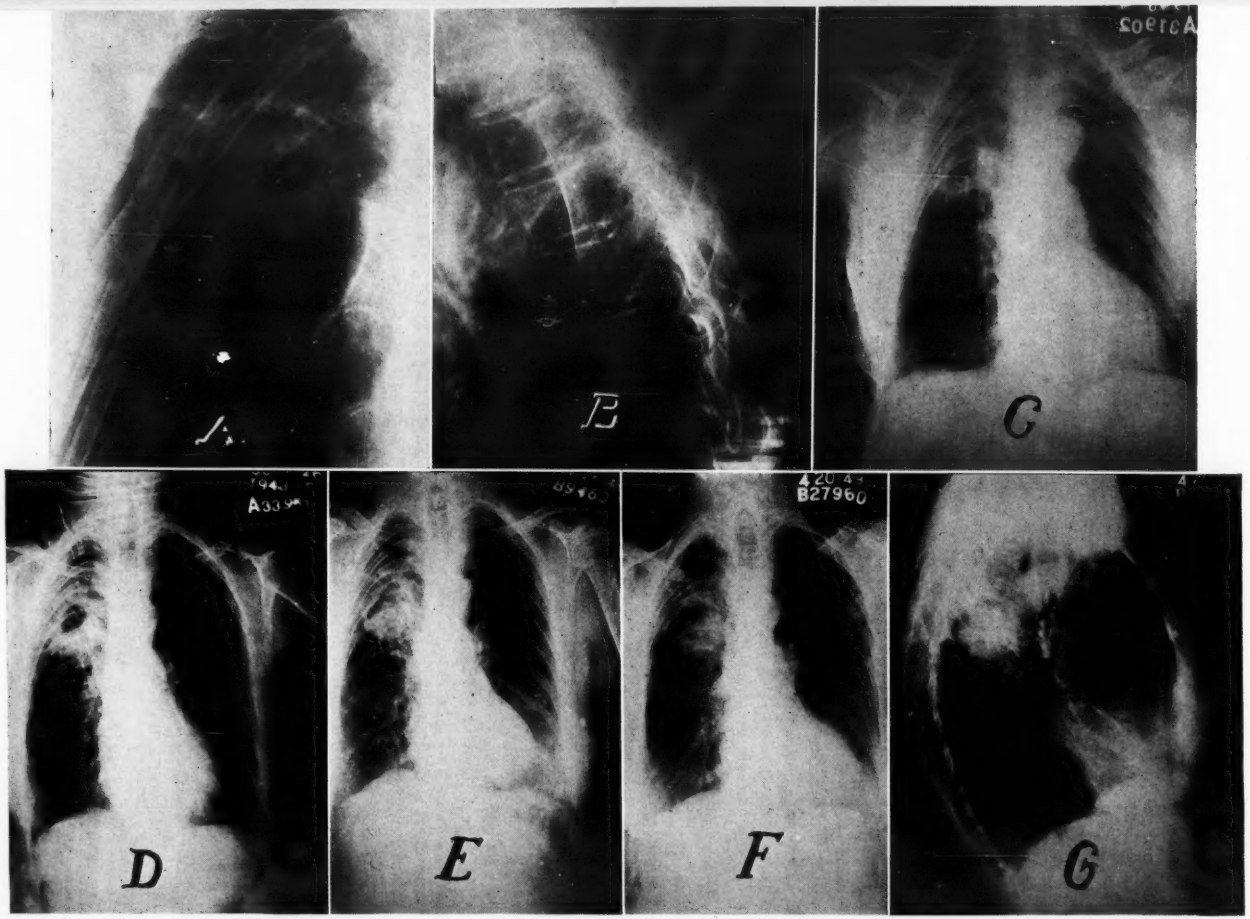

Fig. 1 (Case 1).—Demonstrates the evolution of the air-filled cystic lesion in the right upper lobe into an almost solid tumor mass.
A, and *B*, Roentgenograms taken in April, 1942. A large cystic lesion is present in the right upper lobe.
C, Roentgenograms in August, 1943. There is some surrounding pneumonitis about the cyst.
D, Roentgenogram in October, 1943.
E, Roentgenogram in April, 1948.
F and *G*, Posteroanterior and lateral roentgenograms in April, 1949, show the previously cystic lesion to be almost completely filled in by dense material. No calcification could be demonstrated within this mass.

Roentgenographic examination of the chest showed a well-circumscribed lesion in the apical portion of the right lower lobe. This measured two inches in diameter and had an area of breakdown in the center. No calcification was seen in the mass or in the lung fields.

Direct smears and cultures were positive for tubercle bacilli. Bronchoscopic observation showed the right middle lobe bronchus orifice to be a slit about one-half the usual size. The bronchus was stenosed below this middle lobe orifice.

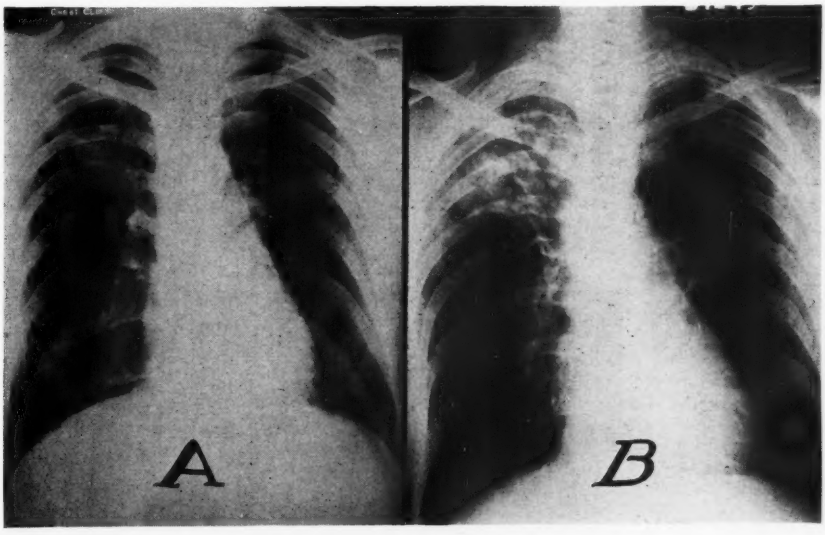

Fig. 2 (Case 2).—*A*, Roentgenogram in July, 1947, showed a circumscribed mass in the right upper lobe. No calcification or areas of breakdown could be demonstrated in this mass. *B*, Roentgenogram on April 4, 1948, demonstrated a diffuse exudating lesion involving the greater portion of the right upper lobe.

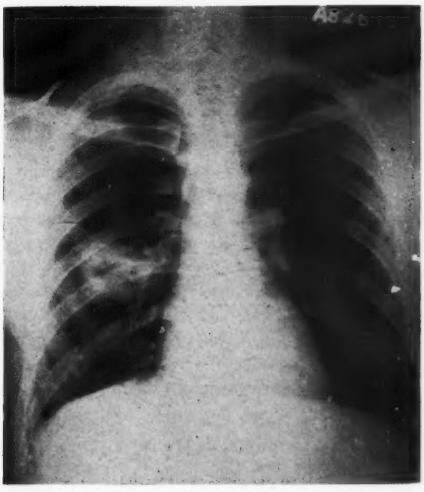

Fig. 3 (Case 3).—Roentgenogram of chest showing right lower lobe mass with breakdown.

g. Roentgenogram in April, 1948.
h and *i*. Posteroanterior and lateral roentgenograms in April, 1949, show the previously cystic lesion to be almost completely filled in by dense material. No calcification could be demonstrated within this mass.

On the fourteenth hospital day a right thoracotomy was done. In the lower and middle lobes there was a hard, nodular tumor which did not have the consistency of a malignant tumor. The lower and middle lobes were resected.

This proved pathologically to be a tuberculoma with cavitation and active tuberculous granulation tissue in the surrounding atelectatic lung. Smears from the central cavity showed a large number of tubercle bacilli.

Discussion.—This case demonstrates a tuberculoma with central cavitation. This tuberculoma was in imminent danger of spread. Despite the positive sputum studies and because of the lack of calcification within the tumor mass, the possibility of a bronchiogenic carcinoma with central breakdown was never excluded. In either case, immediate surgery was the treatment of choice.

CASE 4.—N. S., a 57-year-old white man, was referred by his physician to a clinic in another city because of complaints of parasthesias in both legs. A chest film taken there in 1946 was read as essentially negative. This roentgenogram was reviewed at that clinic in January, 1948, and a question was brought up as to whether there was a tumor mass in the chest, or whether this was merely a soft tissue shadow.

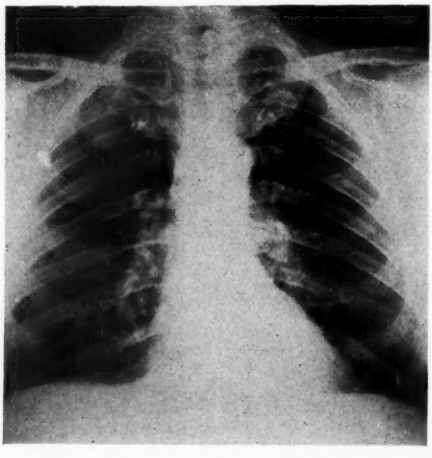

Fig. 4 (Case 4).—Roentgenogram demonstrates an oval mass in right upper lobe. No calcification or breakdown seen.

Because of this questionable finding, a radiographic examination of the chest was done in 1948 which revealed an oval mass 3½ by 3 cm., in the right upper lobe posteriorly. The mass was homogeneous in density. There was no calcification, nor were there any areas of radiolucency in this mass. The previous roentgenograms of the chest which were taken in 1946 were reviewed. The mass in the chest was present at that time, and there had been no change in the size of the mass in the ensuing sixteen months.

Bronchoscopic observation was negative. Cultures and guinea pig inoculations of bronchial aspirated secretions were positive for tubercle bacilli.

It was recommended that this patient have an exploratory thoracotomy; however, he refused surgery.

Discussion.—This lesion is most likely a tuberculoma, and of the type which is liable to undergo spread at any time, although it may remain dormant for years. In view of the tubercle bacilli in the sputum and the lack of calcifi-

cation in the tumor mass, definitive surgery should have been done. However, the patient refused to have any surgical procedures carried out on his chest. It must also be noted that the time interval alone does not rule out a primary neoplastic process. This patient was seen in February, 1950. He was asymptomatic and the chest film was unchanged.

CASE 5.—A. D., a 52-year-old white man, was seen in January, 1949, with a history of a weight loss of twenty-five pounds in the last two years, and a nonproductive cough for six months. Physical examination was essentially negative.

Roentgenographic examination of the chest revealed a solitary rounded mass in the left upper lobe. The outermost border of the lesion was rather indistinct, and seemed to infiltrate the pulmonary fields. There was an area of undiminished density in this tumor mass. There were calcific areas in the hilar nodes.

Bronchoscopic observation was negative. Guinea pig inoculations and cultures from bronchoscopically obtained secretions were positive for tubercle bacilli.

An exploratory thoracotomy was done in March, 1949. There was a tumor mass lying in the upper lobe, apparently arising from the upper lobe main-stem bronchus. It was slightly rubbery in consistency, and there were no outlying regions of metastasis. After study of the pathologic report, the operating surgeon was not sufficiently convinced that the lesion was of neoplastic origin, and he felt that a lobectomy would be a more conservative procedure. If a malignant tumor was found on dissecting the lobe, the surgeon then intended to proceed with the pneumonectomy. On sectioning the lobe, the mass was found to be a typical tuberculoma, and therefore the lower lobe was inflated and the chest wall closed.

The tumor proved pathologically to be a typical diffuse caseated tuberculoma with distinct fibrous encapsulation and with a corona of epithelioid giant-cell tubercles at the periphery. In central portions there was extensive necrobiosis.

Discussion.—The preoperative diagnosis was a pulmonary tuberculoma. In view of the positive culture for tubercle bacilli, and the area of radiolucency in the center of the tumor mass, immediate surgery was the treatment of choice. This patient was seen in March, 1950. He was asymtomatic. No pulmonary complications had developed.

CASE 6.—O. G., a 49-year-old white woman, was hospitalized in September, 1948, because of a mass found in the chest during a routine survey by a mobile chest unit. In 1944, this patient had been treated with intracavitary radium and external radiation for a grade 3 carcinoma of the cervix, with complete regression and no recurrence of the lesion. The patient had no symptoms referable to the chest, and physical examination was essentially negative.

Roentgen examination of the chest showed a small lesion, 2 cm. in its greatest diameter, in the right lower lobe. There was a calcium deposit around the periphery of the lesion. There was a small calcific deposit in the sixth interspace posteriorly on the left. There were multiple areas of calcification in the hilar glands. This lesion conformed to a roentgenographic impression of tuberculoma.

No bacteriologic studies were done.

It was felt by the surgical department that this mass could be a solitary metastasis or a tuberculoma. In view of the patient's excellent health, and because the lesion was in the periphery of the lung, it was decided to admit her for exploration and removal of the mass. An exploratory thoracotomy was done in March, 1949. On entering the chest, a puckered area was seen in the right lower lobe. Since this could not be indentified grossly, it was decided to remove the indurated area. This was sharply dissected out with digital pressure on the surrounding lung parenchyma.

The removed tumor was 2 cm. in diameter, grayish in coloration, and fairly soft in consistency. It was a firmly encapsulated, fibrocaseous, and extremely chalky tuberculoma. No active tuberculous granulation tissue was seen.

Fig. 5 (Case 5).—*A* and *B*, Rounded mass in left upper lobe. The outermost border of this lesion is indistinct and seems to infiltrate the pulmonary fields. There is an area of diminished density within the tumor mass.
C, Photograph of a resected lobe with tuberculoma transected.

Discussion.—There is no doubt that the preoperative diagnosis in this case should have been tuberculoma. Since this lesion was well calcified, and no area of radiolucency could be demonstrated in the tumor mass, it is now felt by this group that surgery was not indicated in this case. The procedure of choice should have been serial roentgenograms at periodic intervals. Patient is at present alive and well, and asymptomatic.

CASE 7.—A. McG., a 49-year-old woman, was hospitalized in January, 1945, because of a mass found in the pulmonary fields in a routine pre-employment examination of the chest. The patient was asymptomatic, and physical examination was negative.

A chest roentgenogram in January, 1945, demonstrated the presence of a solitary, circumscribed, roughly round shadow, 4 cm. in diameter, in the right middle lobe, close to the hilum. This mass was homogeneous in density. Linear calcification was noted along the lower border of the mass; there was also irregular flocculent calcification scattered throughout the mass. There was calcification in the hilar glands.

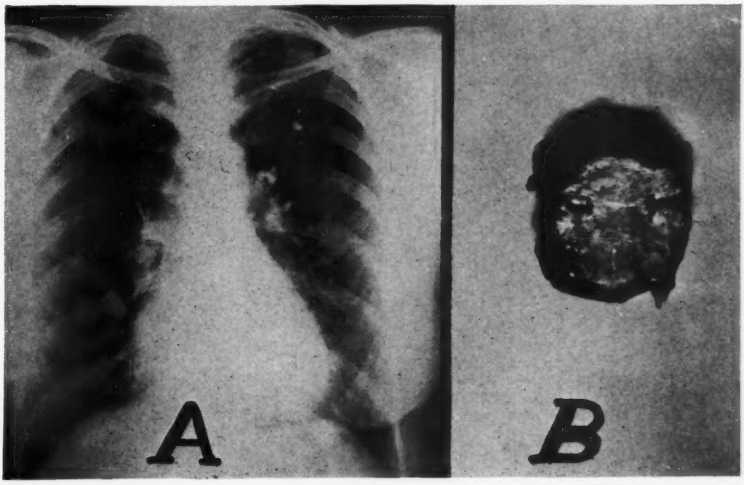

Fig. 6 (Case 6).—*A*, Roentgenogram shows a small lesion in the right lower lobe. There is a calcium deposit around the periphery of the lesion. Calcification can be seen in hilar glands and pulmonary fields.
B, Transected specimen.

Repeated bronchoscopic studies were negative. Bacteriologic studies for tubercle bacilli and fungi were negative. The patient was followed in the Out Patient Department until December, 1948. During this time there was no change in the mass.

On Dec. 12, 1948, when a thoracotomy was performed, a round cream-colored mass was found lying near the hilum of the right lung, half embedded in the parenchyma of the middle lobe. The mass was removed by separating it from the lung tissue.

The specimen was an encapsulated mass 4 cm. in diameter. On section, the tumor was yellowish white in coloration, very firm in consistency, with areas in which there was marked deposition of calcium. The tumor mass appeared to be largely fibrous, with fibers arranged in whorls. In occasional areas the tissue was relatively soft in consistency. The center of the lesion consisted of a conglomeration of old calcified and partly ossified tubercles. These calcified and ossified areas were separated by firmly hyalinized connective tissue which had resorbed part of the calcified periphery of these tubercles. The entire surrounding area was a firm, fibrous stroma comprising a few thin areas of original alveoli.

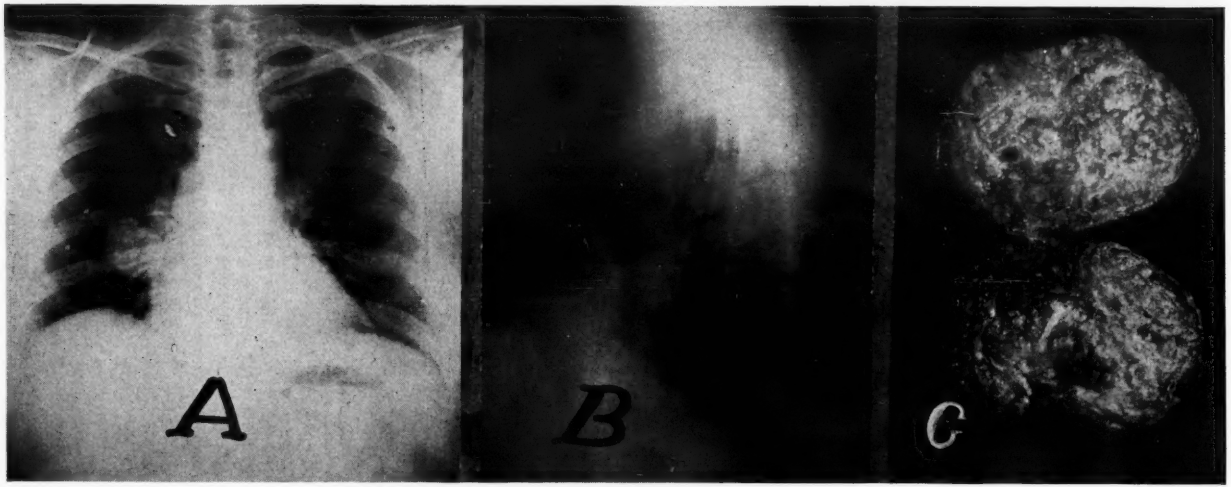

Fig. 7 (Case 7).—*A* and *B*, Posteroanterior and lateral roentgenograms show a circumscribed mass in the right middle lobe close to the hilum. Linear calcification can be noted in the lower border of the mass.
C, Photograph of enucleated tuberculoma.

The patient had an uneventful recovery, and was discharged Dec. 20, 1948. While at home she had a sudden onset of nausea, anorexia, shortness of breath on exertion, and on Jan. 29, 1949, was re-admitted. A right side empyema was discovered, and 300 c.c. of purulent material removed by needle from the chest. On Jan. 30, 1949, an open thoracotomy was done for drainage of the empyema pocket which extended from the apex down to the sixth rib. The empyema cavity was again explored, Feb. 10, 1949, culture of pus from the right chest was positive for tubercle bacilli. The patient improved after this. Roentgenograms showed the empyema cavity to be gradually decreasing in size. Despite the presence of a definite bronchopleural fistula, the wound was completely healed by June, and the patient has since been well.

Discussion.—This case presented a very difficult problem as to the method of treatment. Because there was only a moderate amount of calcification within the mass, a prediction as to the ultimate fate of the tumor could not be made. A thoracotomy was done for this reason. According to the criteria for operation, outlined later in this paper, the surgery in this case might have been deferred and observation continued.

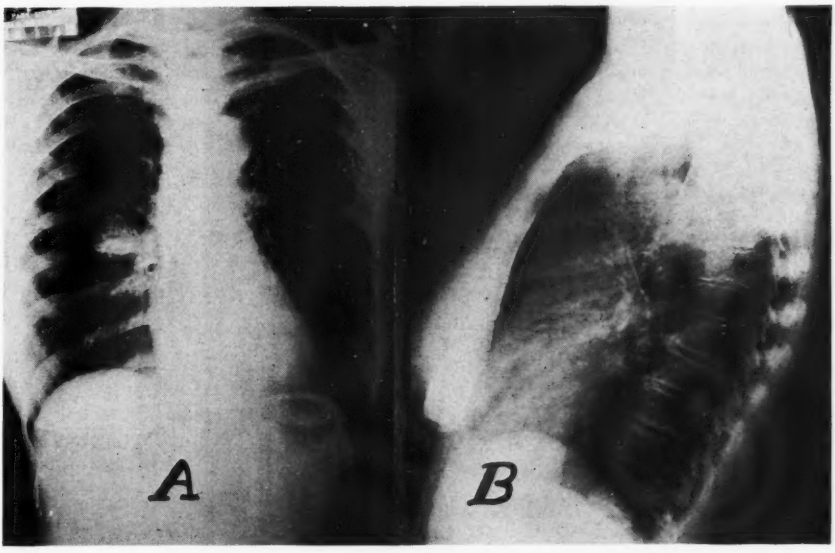

Fig. 8 (Case 8).—*A* and *B*, Roentgenogram shows a singular circular homogeneous density in the right lung, in the angle between the major and minor fissures at the hilum. Multiple areas of calcification are seen in the tumor mass, hilar glands, and lung fields.

CASE 8.—K. C., a 65-year-old woman, was seen in April, 1948, because of the discovery of a mass in the chest at a cancer detection clinic. The patient was asymptomatic, and physical examination was negative.

Roentgen examination of the chest showed a single circular homogeneous density 3 cm. in diameter in the right lung, in the angle between the major and minor fissures at the hilum. There were multiple scattered areas of calcification in the tumor mass, hilar glands, and lung fields.

Bronchoscopy was negative. Repeated sputa and gastric washings on direct and concentrated smears were negative for tubercle bacilli. Smear and cultures from bronchial

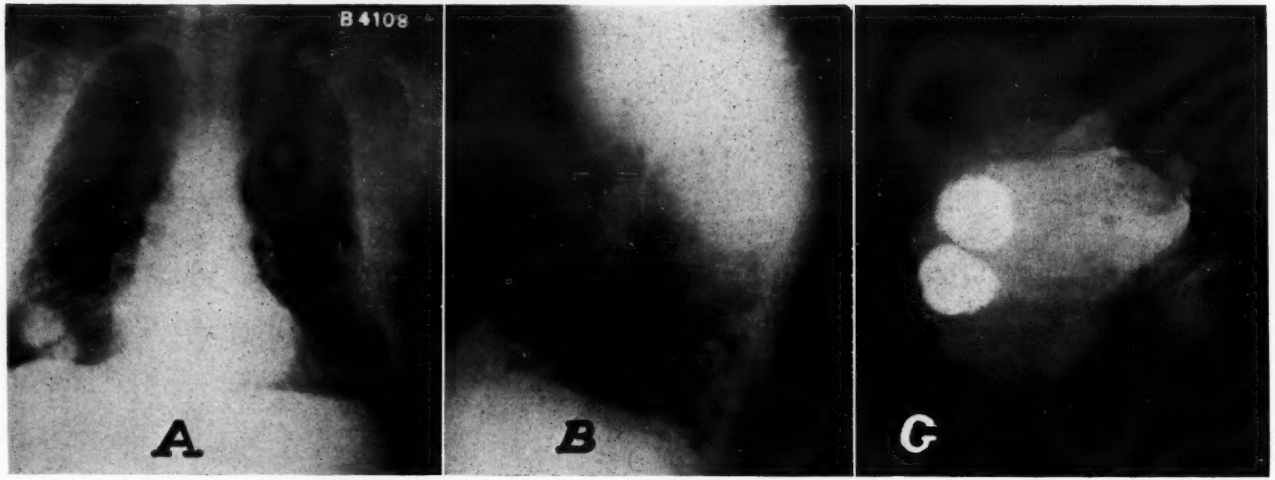

Fig. 9 (Case 9).—*A* and *B*, Roentgenograms in posteroanterior and lateral projections show two rounded lesions in the right lower lobe posteriorly. Calcification is seen in the lower border of the upper tuberculoma. Calcification is seen in hilar nodes.
C, Roentgenogram of resected lobe demonstrates that the tuberculoma contains a large amount of calcium.

aspirations were negative for acid-fast bacilli and fungi. Later cultures and guinea pig inoculations were both reported positive for tuberculosis.

Discussion.—Operation was deemed inadvisable because of the general condition of the patient, repeated negative bronchoscopies, lack of progression of the lesion, and position of the lesion which would have necessitated pneumonectomy. Last chest film was in January, 1950, The appearance was the same.

CASE 9.—C. W., a 54-year-old man, was hospitalized on Jan. 15, 1948. One month before admission he had noticed some shortness of breath on exertion. He had a cough productive of four tablespoons of sputum daily. He also developed a sharp pain in the anterior right chest which lasted one week. Physical examination was negative.

Roentgenographic examination of the chest revealed two rounded lesions in the right lower lobe posteriorly, each being slightly more than 1 inch in diameter, lying one above the other, one slightly anterior. These were round and circumscribed, and in such proximity to each other that they actually overlapped. Tomographic studies of these lesions demonstrated partial calcification in the wall of the superior lesion, and scattered areas of calcification throughout the tumor masses. There were discrete areas of calcification in the hilar nodes.

Bronchoscopy showed a moderate degree of chronic inflammatory reaction. There were no localized lesions in either the right or the left main bronchi, or in the secondary orifices. Bronchial secretions were negative for tubercle bacilli. Cultures were also negative.

On the ninth hospital day a right lobectomy was done. There was found to be an extremely hard white tumor within the substance of the lower lobe, which had the appearance of cartilage on the pleural surface. There were some hard nodes about the lower lobe hilum which were likewise calcified.

On section, there were two, golf-ball size, distinctly encapsulated tumor masses, apparently tuberculomas. One measured 2.8 cm. in diameter, and the other 2.5 cm. Section through the tuberculomas showed a fairly thick fibrous capsule, and extensive calcification of the caseous tissues. The surrounding alveoli were compressed and somewhat atelectatic. No active tuberculous granulative tissue was seen.

On the sixteenth day the patient was discharged with wounds well healed.

Discussion.—The presence of dense calcification in the tumor masses simplified and justified the diagnosis of pulmonary tuberculoma. This calcification was sufficient to be of prognostic significance. Surgery was probably not indicated at this time because of the apparent instability of the lesion as indicated by the amount of calcification within the tuberculoma. Roentgenographic examination of the chest at regular intervals might have been a wiser decision.

CASE 10.—D. N., a 74-year-old white man, was seen in March, 1948, because of an unusual finding in a routine chest film taken by his physician. The patient was asymptomatic on admission, and physical examination of the lungs was negative.

Roentgenographic examination revealed a roughly round tumor, 6 cm. in diameter, extending out from the mediastinum into the lung fields. Lateral film of this chest showed this lesion to be in the middle mediastinum. The mass had irregular borders and discrete areas of calcification. The rest of the lung fields were normal, except for scattered areas of calcification.

Bronchoscopic examination showed the carina to be moderately fixed. The right bronchus was not remarkable. The left bronchus seemed to be pushed anteriorly and upward, but all subdivisions could be recognized. Bacteriologic studies of sputa and bronchial aspirations were negative. Cultures and guinea pig inoculations were negative.

The thoracic surgical consultant felt, because of the bronchoscopic findings, that this case was most likely a lymphoma, and because of the patient's age recommended a therapeutic trial of roentgen therapy before subjecting him to an exploratory thoracotomy. The patient received intensive radiation treatment with no diminution of the size of the tumor.

On Aug. 10, 1948, an exploratory thoracotomy was done. Extending out from the mediastinum into the right upper lobe of the lung there was a mass the size of a small orange studded with calcified pebbles over its surface. It was extremely hard, yellow in color, and did not deform the lung. The operating surgeon felt that this was probably a tuberculoma because of its color, consistency, and surface appearance. Biopsy was not considered advisable. The tumor was not resected because of patient's age and poor general condition. Patient's postoperative course was uneventful.

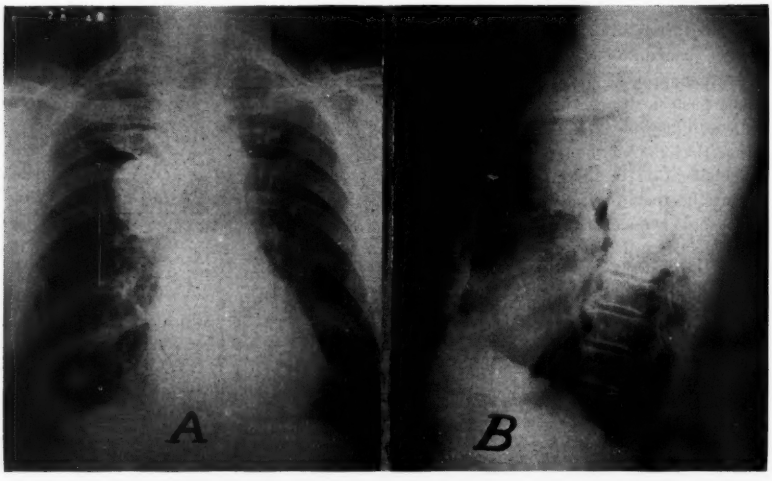

Fig. 10 (Case 10).—*A* and *B*, Roentgenograms in posteroanterior and lateral projections show a roughly round tumor 6 by 6 cm. extending out from the mediastinum into the lung fields. The mass has irregular borders and discrete areas of calcification. Scattered areas of calcification can be seen in the lung fields.

CASE 11.—L. D., a 72-year-old Negro, was seen on June 19, 1946. One year previous to admission a chest plate had been taken, and a diagnosis of *mediastinal tumor with calcification* has been made. On admission to the hospital the patient's chief complaint was marked progressive dyspnea. He had a productive cough, without blood-streaking. At the age of 18 years, cervical tuberculous glands had been removed at the Johns Hopkins Hospital.

Physical Examination.—The patient was a cachectic, elderly Negro. The chest was emphysematous; dullness was elicited over the right interscapular area. Râles and rhonchi were heard in both apices. Remainder of examination was essentially negative. While in the hospital, patient had a daily low-grade fever.

Numerous roentgenograms of the chest were made. Examination of the chest and mediastinum in December, 1946, fluoroscopically and radiographically, showed a huge tumor mass with calcification in its wall. This mass was posterior to the trachea, in front of the spine, and extending to either side of the spine. The trachea was pushed far forward and the bifurcation of the trachea was pushed upward. Multiple areas of calcification were noted in the paratracheal and hilar nodes. Large areas of calcification could be noted in the upper abdomen. This mass was believed to be a posterior mediastinal tumor, with calcification in the wall. The following possibilities were considered: (1) echinococcic cyst; (2) huge tuberculoma with calcification in the wall; (3) nodular enlargement of the aberrant thyroid tissue with secondary calcification.

Repeat roentgenogram in June, 1948, showed infiltration in the right infraclavicular and apical areas, with what appeared to be multiple fine areas of cavitation.

Fig. 11 (Case 11).—Roentgenograms in posteroanterior, lateral, and oblique projections showed a huge tumor mass with calcification in its wall. The trachea was pushed forward and the carina upward. Multiple areas of calcification are seen in the paratracheal and hilar nodes.

Re-examination of the chest on July 3, 1948, showed an increase in the infiltrative lesions, extending down into the right lower lobe from the hilum. There was a small amount of fluid at the costophrenic angle.

Repeat cultures and films were negative for tubercle bacilli.

Discussion.—Cases 10 and 11 are presented to illustrate tuberculomas of the mediastinum. Case 11, in view of the spreading infiltrative lesions in the right upper lobe, is probably a tuberculoma of the mediastinum, although pathological or surgical proof is lacking.

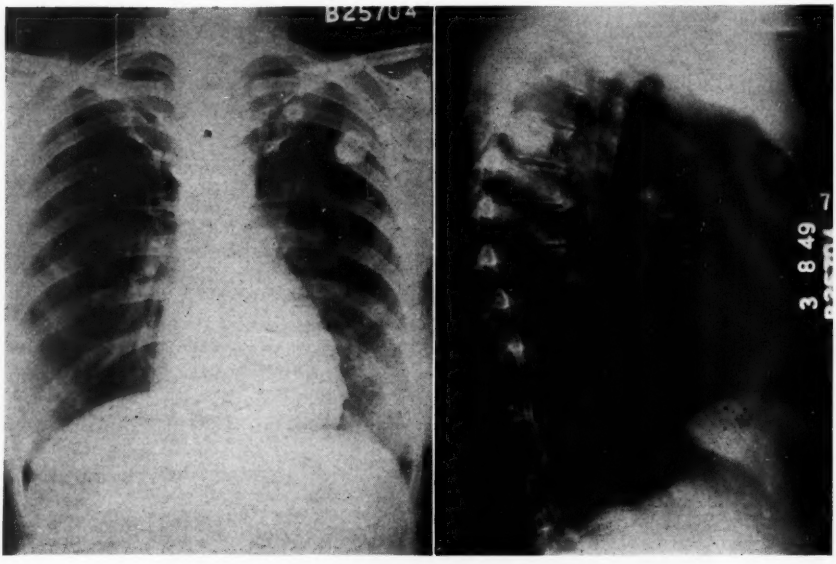

Fig 12.—This case was not presented in the body of the paper. It demonstrates very well two almost completely calcified tuberculomas in the left upper lobe.

MANAGEMENT

The radiographic demonstration of a solitary circumscribed lesion without calcification in the lung fields, in which all other diagnostic studies are negative, demands an exploratory thoracotomy. It is a relatively safe procedure, and has a low mortality and morbidity rate. Since many of these tumors will be malignant, exploration as soon as possible is the treatment of choice. Should the lesion prove to be metastatic, recent reports[25] have indicated that resection of isolated metastatic tumors in the lung fields may be beneficial, particularly if the primary lesion has been eradicated. A large number of these solitary tumors will prove to be of tuberculous origin. There is increasing evidence that resection of localized tuberculous lesions of this type is advisable.

The position of the mass may make the surgical management simple or complicated. The mass, if in peripheral location, may allow easy examination at operation, and, should a tuberculoma or other non-neoplastic process be

present, local resection is feasible and safe. When the lesion is located at the hilum and is not calcified, the matter is much more serious, for, since the majority of such cases will be malignant tumors, we have no alternative but exploration with the idea of pneumonectomy. It is doubtful if sharp local excision of a suspected tuberculoma at the hilum of the lung is good practice. When a local resection of a tuberculoma in the hilar region is done, numerous fine bronchi are opened, whose fluid and air contents are difficult to control. These bronchial contents pass out into the field and invariably contaminate the operative area, increasing the likelihood of a combined infection which may have disastrous consequences for the patient. The one case in this series in which local excision of a hilar tuberculoma was done was complicated by a postoperative mixed empyema and bronchopleural fistula. Therefore, it must be anticipated before surgery that, if the mass is hilar, the surgeon may have to sacrifice the lung, even in the absence of neoplastic disease, since there is often little room for the safe application of less radical measures. Biopsies of these hilar masses are not always safe if the pathologist reports the presence of a chronic granuloma. A peripheral location of the lesion allows more conservatism, if the operator is in doubt, and therefore a greater margin of safety and less physiologic sacrifice for the patient.

The unchanged appearance of the mass in serial roentgenograms over a few months is not convincing evidence that the lesion is benign, as it is precisely this type of slowly growing neoplasm which is often the most favorable for resection and cure. If a tuberculoma can be removed with a wide margin of normal lung by means of a conservative operation, no particular harm has been done. A peripheral lesion without a definite diagnosis demands an exploration. If there seems to be doubt as to the nature of the lesion, and a biopsy does not seem wise, a pneumonectomy should be performed, if sufficient respiratory reserve exists. Should the lesion lie at the hilar region, and ablation by lobectomy is not feasible, and we have the choice of teasing out the the mass from the hilar structures, or pneumonectomy, the latter is probably preferable, although there may be individual factors which would direct the procedure otherwise in selected cases.

Well-calcified tuberculomas without areas of breakdown can probably be considered stable, and it does not seem to the authors that surgical intervention is the best method of management. The incidence of postoperative complications when added to the mortality rate of pneumonectomy, lobectomy, or local excision probably makes operation more hazardous than conservatism in this type of case.

Further observations over a number of years on carefully selected cases must be carried out before the preceding statements can be fully substantiated. However, it is only by this method that we can with reasonable certainty select the proper treatment for each patient.

CONCLUSIONS

1. Tuberculomas may arise from encapsulation of a primary giant tuberculous focus, from encapsulation of a restricted reinfection focus, or by means of a completely blocked tuberculous cavity.

2. The accurate differential diagnosis of a solitary mass within the lung fields may be extremely difficult or impossible, and in a large number of cases an exploratory thoracotomy will be necessary to establish the correct diagnosis.

3. Tuberculomas without calcification, or with areas of breakdown, constitute a potent threat against the continued well-being of the patient, and should be removed.

4. It is probably safe to advise conservative, well-managed observation in patients presenting definitely calcified solitary lesions without any evident breakdown.

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DISCUSSION

COL. JAMES H. FORSEE, Denver.—Added emphasis to the surgical management of tuberculosis may be given by reporting an experience we have had at Fitzsimons in the

surgical management of sixty patients with tuberculoma. Approximately two-thirds of the patients have been handled surgically by wedge excision of the lesion, one-third by lobectomy. We believe this is an actual and potentially dangerous lesion and, in general, that all tuberculomas should be removed by surgical means. True, certain patients who have had for many years known tuberculous lesions, may be treated conservatively.

(Slide.) This illustrates a typical tuberculoma.

(Slide.) This is a surgical specimen—what we call a mature tuberculoma; here is a colored photograph of that specimen.

(Slide.) There is no means with which we are acquainted by which we can tell preoperatively and without examination of pathologic tissue, the true nature of the lesion in most instances. This lesion, as you will note, is circumscribed; it may or may not be a mature lesion; we cannot tell by x-ray findings.

(Slide.) This is the surgical specimen—it is an unripe lesion teeming with tubercle bacilli. The wedge excision which we performed is, as you see, too small for this type of lesion, and had we known beforehand we would have removed more parenchymal tissue.

(Slide.) There is an area of translucency near the border of this circumscribed lesion, which our pathologist, Colonel Mahon, has pointed out occurs in a certain percentage of cases, indicating a break-through of the encapsulated lesion, and we believe it is a definite indication for consideration for surgical excision.

(Slide.) This film again shows a typical tuberculoma, but impossible to tell by roentgenographic examination. This patient did have symptoms of mild fever.

(Slide.) This is the surgical specimen of a lobectomy; the patient had a cavitary lesion, a breakdown of the tuberculoma, and endobronchial disease.

(Slide.) The danger of tuberculoma is well illustrated in this particular instance; a 36-year-old woman who had been watched for seven years. She had been advised to have exploration of this lesion in 1945 in the midst of the war, and she deferred exploration. We saw her in 1947; the tuberculoma had broken down and she had had dissemination of the disease.

In general, calcification takes a long time to develop. Streptomycin has no effect on these lesions and we believe surgical exploration and excision is the proper therapy.

DR. JOHN R. PAINE, Buffalo.—Dr. Culver and I come from the same clinic. Before we get too far out on a limb, I want to say that I personally subscribe to the management expressed by him which, briefly, is this: we think all tuberculomas should be excised except those that are calcified with no evidence of internal breakdown. However, even in that class of patient, we think they should be followed with serial x-ray films every three months. If at any time they do show internal breakdown or radiotranslucency, we think they should be excised.

(Slides.) Dr. Culver's series is twenty-three; this will be his twenty-fourth. This is the last case which has come to our attention. This is the x-ray film of a 51-year-old woman who is asymptomatic; this was a survey film taken last October. We have spent since last November until the first of April trying to find out where the primary tumor was in this case. There are twelve distinct lesions in this woman's x-ray film. On close scrutiny of the original film seven of these lesions showed calcification. Has she got multple tuberculomas? Has she got metastatic carcinoma? We have spent about four months trying to find where the primary tumor might be. Two weeks ago we excised one of these lesions for biopsy.

(Slide.) This proved to be a mature tuberculoma. While it is true that multiple tuberculomas do not occur very frequently, there are three out of the twenty-four. I think these multiple lesions occasionally cause a good deal of doubt as to how they should be treated properly.

DR. EDGAR DAVIS, Washington, D. C.—Several years ago before this Association I discussed a paper on intrapulmonary tumors—I do not recall by whom the paper was given. For a number of years we have been much interested in these solitary tumors. In the original forty cases which I presented, we had a total of 70 per cent malignant and 30 per

cent benign lesions. No tumor was included which showed any evidence of excavation or calcium; nor were tumors included that were located against the diaphragm, the chest wall, or the mediastinum. The average age of the forty patients was 52 years. At that time it was predicted that if a chest x-ray survey could be made whereby a large group of the younger people could be studied, the percentage of malignancies would diminish and there would be a corresponding increase in the so-called tuberculomas. In 1948, a mass chest x-ray survey was carried out in Washington, D. C., at which time about one-half million people above the age of 14 years were x-rayed. We have now accumulated, partly from that mass chest x-ray survey and partly from other sources, a series of 92 or 93 of these solitary tumors. Sixty-seven of these cases have been completely tabulated, of which 55 per cent were malignant. The remaining 25 or 26 cases, although not completely analyzed, are showing approximately the same results. It is interesting to note that of the patients who had no symptoms whatever referable to the chest, 28 per cent were found to be malignant. Although this may be considered a relatively small series, it is sufficient, in my opinion, to justify immediate action and early removal rather than watchful waiting.

DR. W. O. KELLEY, Norwich, Conn.—I agree with Dr. Culver's discussion on the pathogenesis of tuberculoma generally. However, I would like to make the statement that there may be some disagreement as to whether we should actually classify as tuberculomas the formation of an intrapulmonary cavity and later the filling of this cavity with secretion. On the other hand, in tuberculoma, if we can follow the progress, we actually see first a solid tumor, later breaking down to form sometimes a fairly large cavity. To illustrate this I have three cases that could be added to this series.

(Slide.) This is the case of a 23-year-old woman who had three masses, one small, one of medium size, and one large. The patient had a positive sputum which continued positive in spite of pneumothorax treatment.

(Slide.) In the lower mass, the largest one, there is definite rarefaction.

(Slide.) This is the removal of the upper lobe, with an incision made vertically on the apex to the base, showing the first, the second, and the third mass with cavity formation.

(Slide.) This is an x-ray film of another case in which we have proof that there was a tuberculoma; there is probably some breaking down but it does not show clearly here.

(Slide.) The laminagram shows the mass a little more clearly.

(Slide.) This is the resected upper lobe, and here is the tuberculoma in section, one-half here, and one-half here.

(Slide.) Another case with a mass under the right clavicle.

(Slide.) Here is perhaps what we would call a ripened or mature tuberculoma in cross section.

(Slide.) This is the result postoperatively.

DR. DEWITT C. DAUGHTRY, Miami.—This is a very interesting topic. I have recently seen several cases that were of great interest, at least to me. One of these lesions was indistinguishable from a tuberculoma and broke down in a few weeks to produce a disseminated histoplasmosis. Another one was a dense round lesion which was excised, and was a granuloma due to localized blastomycosis. The majority of these lesions that I have seen have proved to be carcinoma. Some of them had not changed in size in two or three years' time. I would like to show one case to demonstrate what may happen to a tuberculoma which had changed very little in five years.

(Slide.) This lesion was present five years ago, asymptomatic, and had not changed until about three months before this film was taken. The patient became symptomatic, had a negative sputum, did not feel well, but did have a cough. The lingula containing the lesion was resected.

(Slide.) This shows that it is a granulomatous tuberculous lesion which has broken down, in spite of the fact that it had apparently been stable for five years.

Seldom can a definite diagnosis be made when we visualize a discrete round solid peripheral opacity on the x-ray film. These lesions must be considered very dangerous, and we should not feel content until we have them in a specimen jar.

DR. W. M. TUTTLE, Detroit.—I do not believe that there is any doubt in the minds of most of us present but what the rounded lesion within the lung should be surgically removed. There is on the other hand a considerable danger because of the fact that many individuals in the field of medicine have come to believe that the rounded lesion is a relatively benign process. The idea is all too current that the rounded lesion can be safely watched, if it does not over a period of months demonstrate growth then it is considered either an inflammatory process or some benign tumor, if it grows then it is considered naturally to be malignant. This, of course, is an absolutely inaccurate view of the facts. There are numerous carcinomas which grow slowly, if at all, over a period of many months. I have recently seen such a lesion in a woman of middle age which was watched for a period of approximately eight months in which no growth was demonstrated. Six months after this watching period she returned with a lesion many times the size of the original one. A pulmonary resection was done and it was evident that the mediastinal glands were involved. The mere fact that an area of calcification is present within the center of the rounded lesion is no proof that the lesion is tuberculous rather than benign. We have recently resected such a lesion in which a carcinoma had developed about a previous Ghon's tubercle. In view of many recent studies which demonstrate that the rounded lesion within the lung carried an incidence of malignancy in from 25 to 50 per cent of the patients seen with this type of lesion it is certainly unsafe to temporize.

DR. JOHN R. MC DONALD, Rochester, Minn.—In a surgical pathological laboratory, between twenty and thirty varying surgical conditions are seen for which resective pulmonary operations are performed. One of the largest groups results from granulomas, there being a great variety that affect the lung. These granulomas may be produced by bacteria, molds, parasites, or chemicals. The histologic pattern is often similar regardless of etiological factors. Too often an opinion is given that a certain granuloma is caused by *Mycobacterium tuberculosis* without any adequate bacteriologic proof. To me it seems unwise to diagnose a given granuloma of the lung as a tuberculoma without this proof.

DR. NORMAN WILSON, Brookline, Mass.—I rise to ask Dr. Culver if he would be kind enough in his closing discussion to tell us from his experience and study, what cases he thinks we should treat as tuberculous patients after the excision. This problem was brought to my mind forcibly by two successive cases which I recently have done, both survey lesions, both having isolated round tuberculomas in the lung tissue, with nothing else seen by x-ray examination or palpation, but with large hilar nodes which looked to be active. I wondered whether he had had this experience, and which patients he felt should be admitted to the sanatorium for treatment.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—I certainly agree with Dr. Tuttle that these rounded tumors, when they are found, should be surgically attacked if there are no special contraindications to operation. I should like to discuss the type of attack that we plan in these cases. Nobody can tell from the x-ray film the microscopic pathology in these tumors. When we explore them, we always make on the table a frozen section from the area of the tumor in order to ascertain what type of pathology is present. If it is a malignant tumor we prefer routinely to do pneumonectomy; only in selected cases where there is good reason for it, lobectomy is done. In the cases where we find it is a benign process, and if this is well localized, we prefer to dissect the pulmonary artery and the main stem of the two veins. We clamp these vessels temporarily. We dissect the bronchus with the bronchial arteries and clamp these as well. We thus get a bloodless field in which we proceed to do a local open excision of the benign process. We pick up and ligate the vessels which are at the bottom of the cavity. We also ligate smaller bronchial luminae, after larger bronchial

defects are sutured, until we have a bloodless field free from air leak also after the clamps are released. The cavity is closed by a few sutures and the chest wall is closed. In suitable cases segmental resection is performed.

DR. CULVER (closing).—It seems necessary to stress the fact that our management of the usual solitary round intrapulmonary lesion is at no variance with the opinions offered by the discussors, as was stated in the body of the paper. Neither do we have any misconception that lack of growth in such a lesion in any way rules out malignancy.

Our main thought has been to stress the point that calcification does not occur in primary pulmonary malignancies.

A tumor without calcification is treated in the accepted manner. It is the calcified lesion without evident breakdown that we treat conservatively. In the older age group, in individuals with diminished respiratory reserve, this conservative management probably is best applied. We feel that the incidence of postoperative complications, when added to the mortality rate of pneumonectomy, lobectomy or local excision, probably makes operation more hazardous than conservatism in this type of case.

This is admittedly a recent survey; however, up to the present time we have encountered no complications in the cases followed. Certainly longer follow-ups will be necessary for final evaluation.

THE HEALING OF SURGICAL WOUNDS OF THE LUNG WITH PARTICULAR REFERENCE TO SEGMENTAL LOBECTOMY

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RESECTION of anatomic segments of pulmonary lobes has become a well-established surgical procedure.[3, 4, 5, 9, 10, 12] The earlier technical reports emphasized closure of the remaining lung defect, usually by a continuous suture.[4, 5] More recently, it has been recommended to leave the raw pulmonary surface as it presents itself following segmental excision.[8-11, 13] With the avoidance of suturing, it was believed that less distortion of the parenchyma resulted, and that better pulmonary function was maintained.

A disturbing feature of this operation is that most surgeons have had a higher postoperative morbidity than after total lobectomies. More complications have occurred no matter how the raw surface of the remaining lobar segment was treated.[2, 5, 10] The causes of the varied types of complications were not always clear, but many authors suspected that the unpleuralized lung surface was the source of trouble.

The fate of the residual lobar parenchyma has been conjectured, but no one has proposed a concept based on direct observation. This is due to scanty autopsy material since the mortality of these cases is low. It also has been rare for a surgeon to examine the results of a segmental lobectomy at a subsequent operation.

The following investigation was performed to determine the sequence of pathologic events in the healing of a parenchymal wound following the removal of an anatomic segment of lung. In most instances a block excision of an adjacent lobe without regard for intersegmental planes was also performed for comparison.

METHOD

Large adult cats were chosen for this study. After nembutal narcosis, a small catheter was inserted into the trachea and ether anesthesia was started. The pleural cavity was entered through the seventh intercostal space. In most instances the apical segment of the lower lobe of the right lung was chosen for resection.

The pulmonary lobes always had complete fissures. The apical bronchus of the lower lobe was identified and isolated for a short distance. A fine silk suture was tied about it

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close to the stem bronchus to the lower lobe. The bronchus was divided between this tie and a clamp placed distally. With slight traction on the clamp, the segmental vein and artery were readily seen and divided between ligatures. The intersegmental plane was identified with gentle blunt dissection near the clamped bronchial end, and the segment was removed with continuance of this technique. This maneuver was performed with ease and no troublesome bleeding from intersegmental veins was encountered. The resultant defect was usually smooth surfaced and measured approximately 2 by 1.5 by 1.5 cm. There was practically no capillary ooze from the surface, and alveolar leaks were either transient or absent. Increased intratracheal pressure demonstrated usually an airtight pulmonary wound. No attempt was made to pleuralize the defect.

The middle lobe was rotated into the wound and its tip was amputated with scissors. Bleeding ceased after the application of warm moist pads for several minutes. At first, the operation was terminated leaving this lesion unsutured. However, one animal died of empyema and a bronchopleural fistula from this defect. In subsequent cases, therefore, the cut end of bronchus was closed with a single mattress suture of fine silk. In most of these animals, the healing progress of a small parietal pleural wound also was studied and the findings will be reported later. The chest wall was closed in layers with silk about a small catheter. Near the end of operation, as much air as possible was aspirated from the chest through the catheter which was then removed. No dressings were applied and the animal was returned to its cage.

The postoperative recoveries were usually uneventful. No roentgenograms of the chest were taken and no thoracenteses were done. None of the animals in this series showed signs or symptoms of infection and all appeared healthy at the time of sacrifice. They were given lethal doses of ether at intervals from two days to six weeks after surgery. The abdomen was opened, and after the viscera were examined, they were retracted caudad. The pleural cavity on the operated side was first entered through an anterior diaphragmatic incision. In this manner the amount of residual pneumothorax could be evaluated easily. When this was determined the anterior chest wall was removed en bloc so that all the thoracic structures could be viewed in situ. With gentle manipulation of the wounded lung, pleural adhesions, the state of the wound, and possible pulmonary distortion were noted.

The lungs were removed and inflated with Bouin's solution via the main bronchi. The specimens were placed in this solution following ligation of the bronchi. Sections were taken from normal and wounded parts of the lungs. These were prepared with hematoxylin and eosin and Masson's trichrom stains.

RESULTS

Gross.—Most animals killed within the first six days after operation had minimal pneumothoraces. One cat was sacrificed on the second day because of moderate respiratory embarrassment. It had a marked bilateral pneumothorax that was caused by a leak from the cut end of the middle lobe branch bronchus. In this instance, the bronchial suture did not prevent a bronchopleural fistula.

Postoperative pleural effusions were not remarkable. There were small amounts of serosanguineous fluid in the costophrenic angles during the first five days. Even this was not observed later except in the cat killed at the end of three weeks. The pleura was always smooth and glistening.

One of the animals killed on the second day had fibrinous adhesions between the middle lobe defect of the right lung and the adjacent pericardium. The wound of the segmental resection of the right lower lobe remained free from adhesions until the sixth postoperative day. During the subsequent period it usually became adherent to the overlying upper lobe or to the pleural scar of the chest wall incision. The density of adhesions varied. When there was symphysis of the defect and the upper lobe, no attempt was made to separate them and both lobes were included in the sections. At no time was there gross distortion of the lung.

The middle lobe defect following amputation of its tip was puckered by the placement of the peribronchial suture. It was difficult, therefore, to determine its dimensions accurately. Precise measurements of the lower lobe segmental lesion could be made unless it was firmly adherent to adjacent structures. It did not show any change in size until the ninth day when it began to contract progressively. By the third week the center of the wound was depressed and its borders were elevated and rounded. The segmental lesion was covered by a thin layer of blood clot up to the third postoperative day. From the fourth to the seventh day, it was overspread with a film of fibrin. Thereafter, the surface had fibrous characteristics.

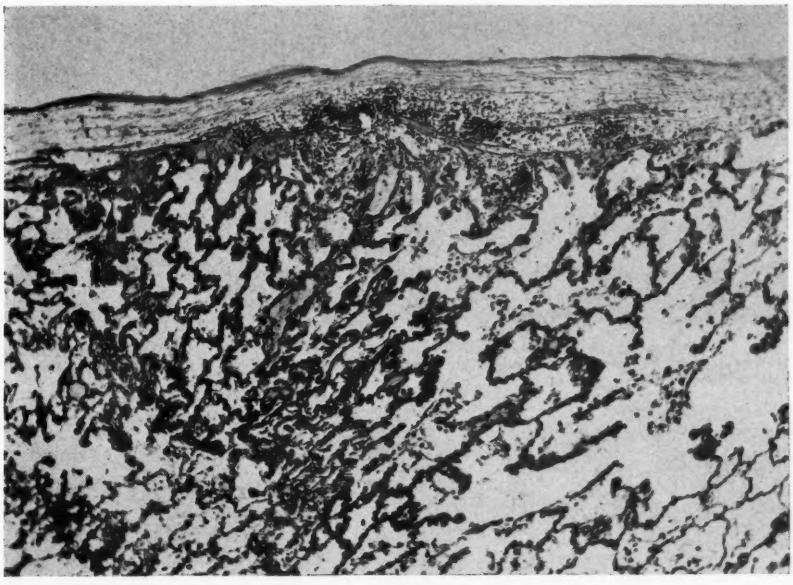

Fig. 1.—Wound of lung two days after segmental lobectomy. Note partial aeration of alveoli and increased caliber of capillaries (×139).

Microscopic.—The sections taken from the cat killed on the second postoperative day were the earliest studied by microscopy. The upper lobe of the right lung and lower lobe of the left lung showed evidence of patchy atelectasis. The bronchi in the involved sections contained considerable amounts of exudate. The segmental lesion of the lower lobe of the right lung was covered with a thin layer of blood clot (Fig. 1). The air sacs subjacent to it were aerated but smaller than elsewhere. In most places septal walls formed the border of the wound. Inflammatory cells near the injury were few and polymorphonuclear leucocytes were absent. The bronchi in these sections were free from blood, exudate, and retained secretion.

Sections from the cat killed three days after operation showed all pulmonary lobes to be well aerated. The segmental lesion was overspread by laminated strands of fibrin with intervening red blood cells in rouleaux (Fig. 2). Again there was a paucity of inflammatory cells. In one portion of the wound, there was evidence that the intersegmental plane was transgressed with resultant hemorrhage into the parenchyma and attenuated alveolar capillaries. This suggested a diminished blood flow through the area. The sections of this lesion showed air in all the alveoli, but there was pulmonary edema near the wound.

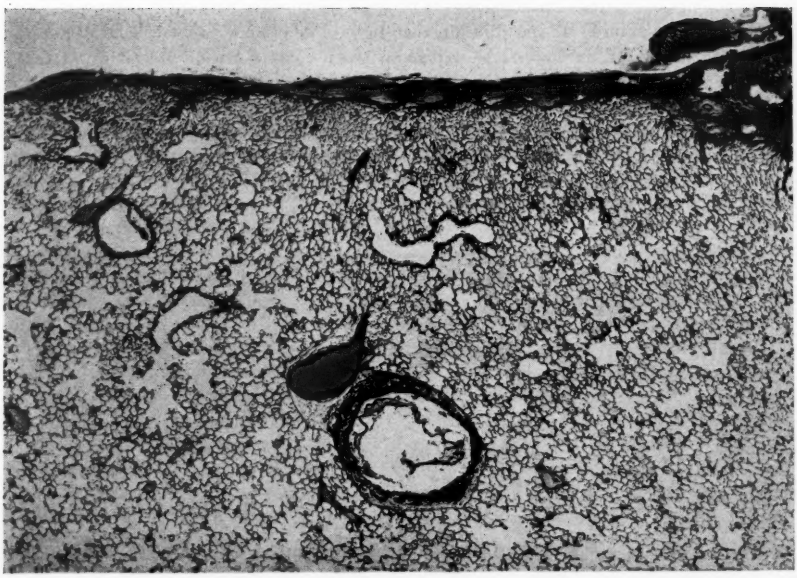

Fig. 2.—Segmental wound three days after operation. At the right the intersegmental plane was crossed, causing hemorrhage into the parenchyma locally. The remainder of the lobe is well aerated. There is pulmonary edema, especially near the wound (×18).

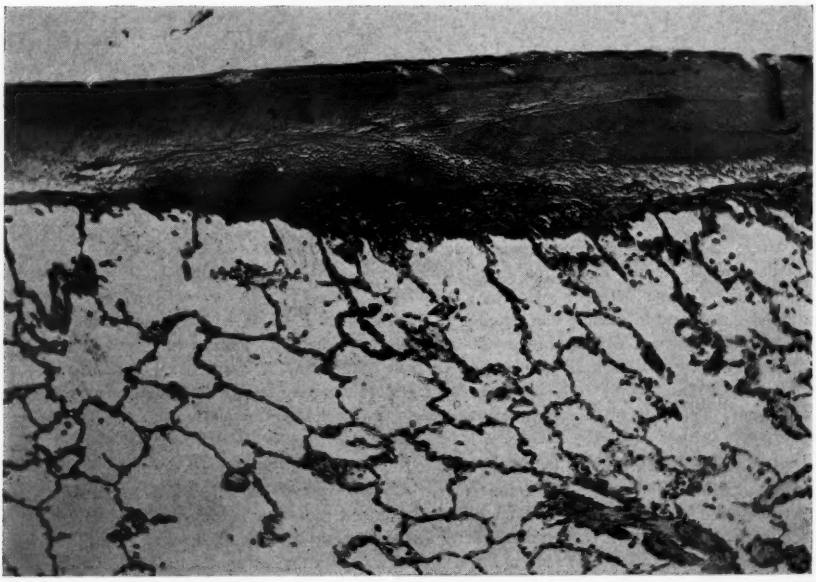

Fig. 3.—Segmental wound four days after operation. Pulmonary aeration adjacent to the wound is more complete. The lung border of the wound is formed by alveolar walls (×139).

During the next two days definite trends occurred. The partially excised lobes showed pulmonary edema. The lungs were well aerated except the middle and lower lobes on the right which had patchy areas of atelectasis, especially near the lesions. On the fifth day, the segmental wound was covered by a fibrinous layer containing disintegrating red blood cells. The layer was much thicker than noted on the previous days and it extended for a considerable distance over the adjacent pleura. Only directly over the wound did it contain all types of inflammatory cells in moderate numbers. Fibroblasts from the wounded lung invaded the fibrin in a patchy manner. Aeration of the underlying lung was variable in different sections but it was never completely atelectatic. The alveolar lining cells near the lesion were large and prominent. A bronchus in this lobe contained exudate.

Fig. 4.—Segmental wound six days postoperatively. The wounded lobe in the lower one-half of the picture is adherent to the upper lobe by fibrinous agglutination. The wound is covered by a fibrotic layer (×31). (See text.)

The sixth day segmental wound was agglutinated by fibrin to the adjacent upper lobe (Fig. 4). The wound was covered with a layer of fibroblasts that paralleled its surface, but they did not extend to the adherent pleura of the upper lobe. Inflammatory cells were rare. Although the parenchyma showed normal aeration, some alveoli contained edema fluid. The bronchi in all sections were clean.

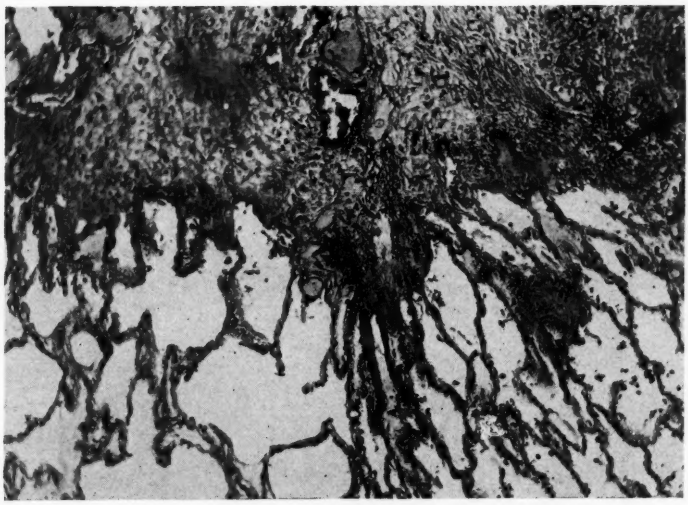

Fig. 5.—Segmental wound eleven days after operation. Note fibroblastic invasion of the fibrin layer and capillaries in the wound (×139).

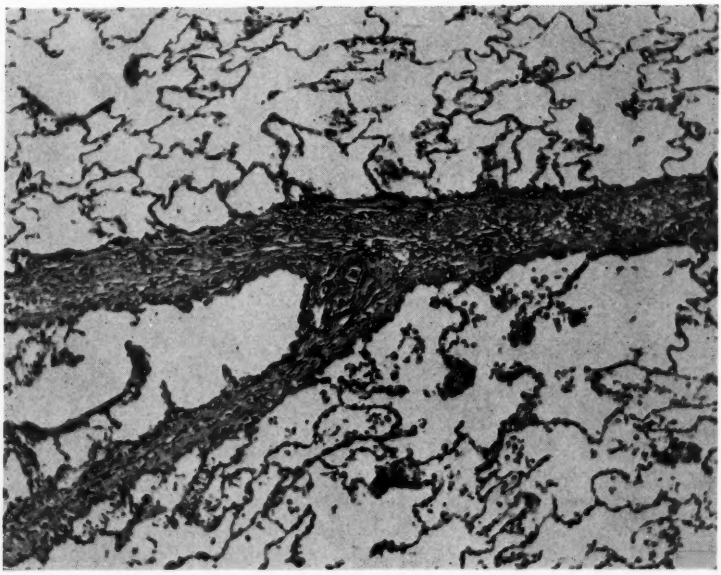

Fig. 6.—Segmental wound three weeks after operation. The wounded segment is in the lower one-half of the picture. Fibrosis has extended along the interlobular septum (×139).

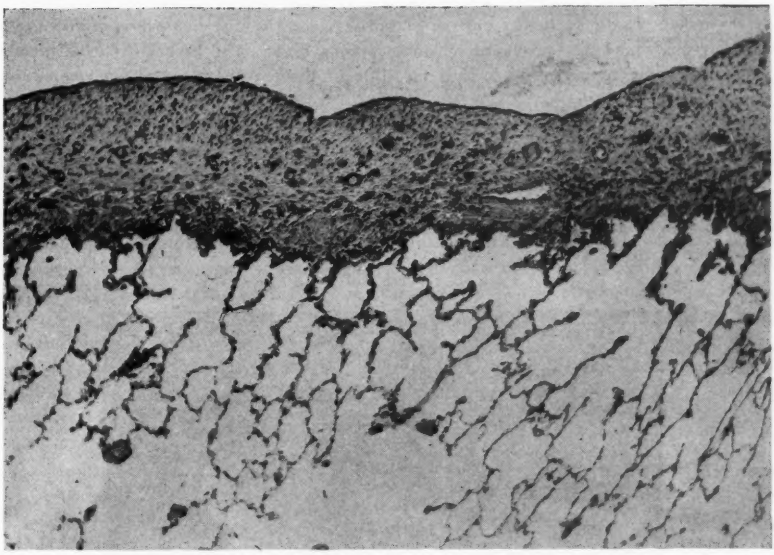

Fig. 7.—Segmental wound six weeks after operation. The scar is mature, undergoing hyalinization, and it contains numerous capillaries. The surface is covered by a single layer of cells (×139).

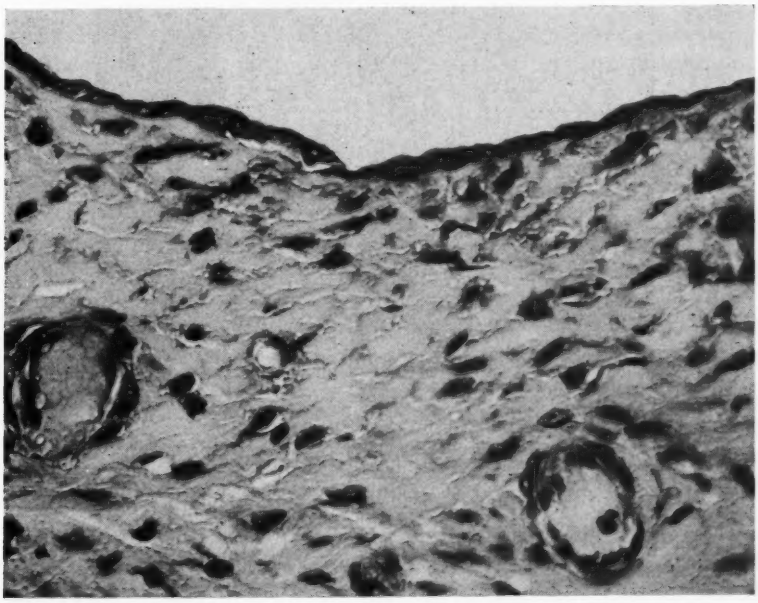

Fig. 8.—Higher magnification of Fig. 7 to demonstrate the cells on the wound surface (×1,064).

There was little noteworthy change until the ninth postoperative day. All lobes were fully expanded. The nonadherent portion of the segmental wound was overlaid with acellular fibrin that was completely enveloped by dense fibrous tissue. The bronchus to the remaining segment was damaged by suture so that the state of the parenchyma was not comparable to previous sections.

The next animal was killed on the eleventh day. There was a fibrotic symphysis of the segmental lesion and overlying upper lobe. A few clumps of fibrin were enmeshed in the adhesion but these were being invaded by fibroblasts (Fig. 5). Inflammatory reaction was minimal and the fibrotic area was well vascularized. The parenchymal pattern adjacent to the wound was slightly irregular but well aerated. The bronchi contained moderate amounts of exudate and there was peribronchial edema.

Fig. 9.—Defect following amputation of the tip of the right middle lobe six days after surgery. The bronchus was closed by a mattress suture. Note the parenchymal degeneration, distortion, and endobronchial exudate (×31).

The animal killed at two weeks was different from the rest because the branch of the pulmonary artery to the lower lobe was ligated. The pleura contained dilated blood and lymphatic vessels. There were scattered small scars throughout the lower lobe, and fibrosis in the wound was extensive. The wound contained several small abscesses.

The changes between the eleventh day and third week were minimal. At four weeks the scar of the segmental wound was mature, contracted, and thinner. There was slight parenchymal distortion due to contraction of the scar. Some endobronchial exudate in the wounded lobe was noted.

By the end of the sixth week, the wound was covered by a thin band of fibrous tissue that was undergoing hyalinization (Fig. 7). Many capillaries were present throughout the scar. In places it was covered by a single layer of cells that had many characteristics of adjacent pleural cells (Fig. 8). The interlobular septa were thickened by fibrous tissue which extended from the surface scar. A disturbing feature was the presence of plugs of exudate in some of the bronchi. Close to the bronchi were small abscesses and areas of pneumonia. It could not be ascertained whether this infection was incidental or secondary to the defect.

Following amputation of a portion of the middle lobe and closure of the open bronchus by suture, wound healing progressed in a characteristic manner. On the second postoperative day, the parenchyma was normal except for distortion near the bronchial suture. From this point, the alveolar walls radiated out in every direction. The alveoli were elongated and flattened near the suture. Between the wound and the suture, there was considerable hemorrhage into the parenchyma, deposits of fibrin, and invasion of all types of inflammatory cells, especially polymorphonuclear leucocytes. On the following day the capillaries near the wound were engorged and the inflammatory elements were greatly increased.

On the fifth day the middle lobe defect peripheral to the suture consisted of degenerating, airless parenchyma. There was active fibroplasia throughout the damaged area and this activity extended over the adjacent pleural surfaces. In the damaged area of the wound six days old, there were numerous clefts that represented intact alveoli caught in the organization (Fig. 9). Thereafter, organization of the wound and the adjacent distorted lung proceeded rapidly. By the eleventh day the surface of the lesion was invested by a dense fibrous layer. Fibrosis elsewhere was diffuse and less compact.

DISCUSSION

This study was designed primarily to determine the healing process of a lung wound following segmental resection in the cat. The portion removed was usually the apical segment of the right lower lobe. Although the chest wounds of these animals were closed without drainage, they had small postoperative pleural effusions. There was no demonstrable escape of air from the segmental wound. Because of residual pneumothoraces, few adhesions were noted during the days immediately after operation. After the sixth day, however, the wound usually was either adherent to the adjacent lobe or to the thoracotomy wound. The wound surface was invariably smooth despite microscopic evidence that the intersegmental plane was crossed occasionally at operation. Gross contraction of the wound was not observed until the ninth postoperative day. At no time was there gross distortion of the injured pulmonary lobes.

By microscopy, healing was ideal when only the anatomic segment was resected. Injury of the pulmonary artery or bronchus to the unpleuralized segment had a detrimental effect on wound healing and the adjacent parenchyma. There was evidence of local parenchymal ischemia and intra-alveolar hemorrhage where the intersegmental plane was crossed (Fig. 2). All of these digressions caused a significant inflammatory response.

Under ideal conditions healing progressed in the presence of few inflammatory cells. Polymorphonuclear leucocytes were virtually absent. The lesion was first covered by a film of blood clot which gradually became amorphous and thicker during the first postoperative week (Fig. 3). Fibroplasia was not active

until the fifth day. At this time fibroblasts grew from the lung substance into the overlying film in a patchy manner. On the following day, the wounded lung was completely covered by these cells (Fig. 4). With contraction of the wound, there was apparent radiation of the alveoli from the surface of the lesion. New capillary formation in the wound covering was definitely evident on the eleventh day (Fig. 5). By the end of the fourth week, the scar was mature. From the third to the sixth week, it became progressively thinner. At six weeks, it was covered by a single layer of cells that were similar to the adjacent pleural cells (Figs. 7 and 8). The possibility that they were fibroblasts arising from the scar could not be excluded. Joannides and associates[6] thought that lung wounds ultimately were covered with endothelial cells.

When the dissection proceeded as planned, the border of the lung beneath the film was formed by alveolar walls (Figs. 1 to 3). During the first five postoperative days, the parenchyma subjacent to the wound was aerated but the alveolar caliber was often not as large as elsewhere (Figs. 1 and 2). This was partially due to widening of the septa. There were patchy areas of atelectasis in the injured lobes during the first five postoperative days. This pathology was seen in unoperated lobes only on the second day. Usually atelectasis was associated with collections of endobronchial exudate in the same sections. After the sixth day, this combination was not observed, and pulmonary aeration was complete. Pulmonary edema in the wounded lobes was present up to the fourth day when it was maximal. It gradually decreased thereafter.

Fibrosis of the lung was absent if there was no alveolar damage. With parenchymal injury, inflammatory changes ensued and fibrosis was eventually considerable. Microscopically, distortion of the lung was not an early manifestation. It occurred later with contraction of the wound and fibrous thickening of the interlobular septa (Fig. 6). Damage to the lung, however, was always followed by distortion of the alveolar pattern. The presence of endobronchial exudate four and six weeks after operation was disquieting. It was associated with peribronchial inflammation and small areas of pulmonary suppuration. From these limited observations it could not be determined whether this pathology resulted from diminished local resistance of the operated lobes.

The comparison of the segmental wound and middle lobe wound following amputation of its tip is not entirely applicable since a bronchial suture was used in the latter. However, the combination of nonanatomic excision and suture caused a reaction far different than was seen after segmental resection. There was considerable strangulation and death of pulmonary parenchyma (Fig. 9). Inflammatory reaction was extensive and polymorphonuclear leucocytes were numerous. Fibroplasia occurred earlier and it was more profuse. Greater distortion resulted from the fibrosis, and the "strand pattern" described by Joannides and associates[6] was evident about the stitch. This type of wound, however, had no demonstrable effect on the lung proximal to the suture. The only bronchopleural fistula in this series resulted from one of these middle lobe excisions even though the cut end of bronchus was sutured.

Segmental resection was easier to perform in these animals than in human beings requiring the operation. This is understandable since a normal pulmonary segment in the human being is usually easier to excise than a pathologic segment. The lungs of the cats in this experiment were normal and the intersegmental divisions were found with facility. In spite of this, it was evident by microscopy that the intersegmental plane was crossed on several occasions. It may be assumed, therefore, that greater parenchymal damage of the remaining segment will occur during anatomic segmental resection for pulmonary disease. This is probably the cause of persistent alveolar and small bronchial leaks. If the removal of a diseased segment is difficult because of fibrosis at the intersegmental plane, the procedure probably should be changed to include an additional segment or to a total lobectomy.[7] On the other hand, when an anatomic segment can be removed with minimal parenchymal damage to the remaining segment, the lung wound will heal well without pleuralization. From the evidence presented here, approximation of the pleural edges by suture probably will cause unnecessary tissue destruction with subsequent local inflammation, fibrosis, and possibly infection.

CONCLUSIONS

In the experimental animal, a precise anatomic resection of a pulmonary lobar segment causes a wound in the lung that heals ideally. A mature surface scar develops over the course of four weeks with minimal distortion of the parenchyma. If lung tissue of the remaining segment is damaged during operative dissection or if sutures are taken, local fibrosis and distortion results. Occlusion of the bronchus or pulmonary artery to the wounded segment also alters healing. Following resection of an anatomic segment of a lobe, it is unnecessary and probably unwise to pleuralize the surface of the lung wound. It is suggested that segmental lobar resection in human beings be abandoned for an alternative procedure if the operator cannot dissect easily in the intersegmental plane. Many postoperative complications of segmental excision are related probably to undue trauma and residual disease at the line of resection.

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NOTICE

Applications for Membership

Applications for membership in the American Association for Thoracic Surgery must be sent to Dr. B. Noland Carter, Cincinnati General Hospital, Cincinnati, Ohio, before January 1, 1951.